

**ANESTHESIA FOR
SURGERY OF THE HEART**

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Anesthesia for Surgery of the Heart

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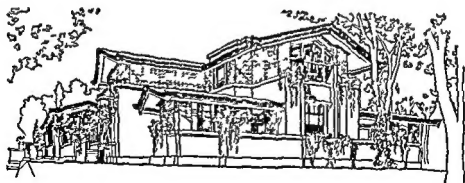
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DEDICATION

*To my wife
Helen Jane Keown*

FOREWORD

THIS PRESENTATION is a milestone in the development of cardio-anesthesiology. It represents diligent research the results of which have been eminently gratifying. Its timeliness is attested by the rapid expansion of the cardiac surgical field and the concurrent need for especially qualified anesthesiologists.

It is fitting that this should be a summarization of the work of my good friend and close associate Doctor Kenneth Keown who has pioneered this specialty. The competence of the anesthesiologist is responsible in large part for the tremendous inroads made in cardiac surgery and without his significant contributions the operative mortality and morbidity which we have encountered would have been considerably greater.

It is only right that his great knowledge and understanding should be disseminated widely that others may avail themselves accordingly.

C P BAILEY, M D

PREFACE

THIS WORK represents an attempt to present a handbook for the student of anesthesiology who deals with anesthesia for surgery to correct both congenital and acquired cardiac abnormalities. The essential symptoms, physical findings, and laboratory results for each of the lesions for which surgery is now considered applicable have been outlined in separate chapters.

It has been my desire to outline the selection of patients, preliminary medication and anesthetic management in a manner that is not controversial, but applicable wherever an anesthesiologist practices medicine.

However, I have made no attempt to include a detailed step by step description of techniques for anesthetic management believing cookbook recipes to be a poor way to teach. Case reports have not been included, as I feel little but words would be added to the manuscript.

Concepts of approved medical practice will vary in the future from those we now consider valid, and no essay on anesthesiology for cardiac surgery can be considered final. This represents the knowledge gained after ten years spent anesthetizing patients for heart surgery in one of the pioneering cardiovascular centers in the United States.

ACKNOWLEDGMENTS

IT IS WITH PLEASURE and deep gratitude that I make the following acknowledgments

To Mary Lou Buckley, M D , and William A Weiss, M D , for their aid in clinical anesthesiology for cardiovascular surgery

To Charles P Bailey, M D and his surgical associates, Houck E Bolton, M D , and Henry T Nichols, M D who have been a constant source of inspiration through the many advances made by them in cardiac surgery

To the cardiologists of the Bailey Thoracic Clinic who have been most helpful in the preparation of the manuscript in particular to William Likoff M D , Senior Cardiologist, who has proofread the manuscript, making helpful suggestions where they were indicated

To Daniel F Downing, M D the pediatric cardiologist of Hahnemann Hospital Philadelphia who has read the chapters on congenital defects and offered many helpful suggestions

To Harry Goldberg M D , our clinical cardiophysiolgist who has given me aid in the preparation of the data obtained from cardiac catheterization

To the many resident anesthesiologists who have offered their time and effort so willingly to this newest field in anesthesiology among them Charles W Hoyt, M D Philip Boyer M D , Loftus Hengeveld M D , and David Mendelsohn M D who were particularly interested in anesthesia for cardiac surgery To Richard Albany for his illustrations To my faithful secretary, Frijs Anne Eaton, without whose help this manuscript could not have been completed especial acknowledgment is gratefully made

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**ANESTHESIA FOR
SURGERY OF THE HEART**

INTRODUCTION

THE SUCCESS of surgery for heart disease is a direct result of six things (1) anesthetic developments that allow thoracotomies to be successfully accomplished (2) the development of chemotherapeutic agents that will minimize or prevent postoperative infection, (3) the development of modern blood banks which allow blood to be stored and used as needed during surgery, (4) the ability of the cardiovascular surgeon to solve the mechanical problems of the various heart lesions (5) the desire of the practitioner of medicine to aid his patient, and (6) the courage manifested by the patient physician, surgeon and anesthesiologist in attempting the unknown and uncharted course

No one single factor has been all important in the achievement of success in this newest field of medicine. It is truly the teamwork of the entire medical profession, nursing personnel, and hospital staff that has afforded the patient a new tenure on life.

From ancient times to the present, infants, children and adults have been prey to the ravages of heart disease. We now believe the portal is open and that the knowledge of our predecessors in anesthesiology and recent advances of our own will greatly aid in the curtailment of death due to heart disease.

Meltzer and Auer made their classical contribution on *Respiration without Respiratory Movements* in 1909, and should be remembered as the forerunners of endotracheal insufflation. In 1913, Chevalier Jackson published an article intended for anesthetists, strongly advocating direct vision laryngoscopy prior to intubation.

Magill and Rowbotham, during World War I, introduced large bore catheters into trachea and were the leaders in the abandonment of the insufflation technique of Meltzer and Auer in allowing the respiratory cycle to be consummated through a single large bore endotracheal tube.

Ralph Waters applied to clinical anesthesia the principles of the absorption of expired carbon dioxide by chemical means which had also been advocated by Snow, Kuhn and Dennis Jackson.

Tuffier, in 1913, was the first to attempt corrective heart surgery on a human patient. His patient had aortic stenosis and his procedure must be considered successful as the patient lived for several years following the intervention. As stated previously, rhythmical inflation and deflation of the lungs by means of an endotracheal catheter was employed.

Doyen, in 1913, deliberately attempted to open a stenotic pulmonary valve by inserting a cutting edge instrument into the right ventricle and out the pulmonary artery. These two interventions in the same year on humans opened the way to serious cardiac surgery.

In Boston, in 1920, Levine and Beck began to investigate the surgical approach to mitral stenosis, and in 1923 operated on their first case.

Souttar, in 1925, inserted his finger into the mitral valve of a patient to relieve the stenosis by digitally dilating and splitting the commissure. The patient succumbed five years later as a result of a cerebral embolization.

HISTORY

EARLY WRITINGS about anesthesia for heart surgery are practically non existent. It is extremely interesting to review the history of medicine as applicable to the necessity of an artificial airway for the maintenance of life when the thorax is open.

Paracelsus in the Sixteen Century inserted a tube connected to a bellows into the mouth of an asphyxiated patient. Andreas Vesalius was the first to deliberately intubate the trachea of an animal to overcome the lethal effects of a pneumothorax. Robert Hooke in 1667 presented a paper before the Royal Society of London describing a technique by which the trachea could be cannulated and life preserved in animals by blowing air into their lungs with a bellows. John Snow was certainly the first to administer an anesthetic agent by means of inhalation through an endotracheal tube. This was accomplished in 1858. It was William Macewen of Glasgow who used endotracheal anesthesia as we know it by inserting the endotracheal cannula through the glottic opening.

In 1869 Trendelenberg was the first physician to describe an inflatable cuff around an endotracheal cannula to secure an air tight fit against the trachea.

Maydl of Prague and Eisenmenger of Vienna both published in 1893 a description of a technique for the administration of anesthesia by means of cannulation of the trachea. Tuffier and Hallion in Paris successfully opened the thorax in animals when the trachea was intubated and ventilation was secured by means of bellows. They were also the first to use rhythmic inflation of the lungs.

of this writing Harken reported his first success in the same year

Other advances followed rapidly Sellors and Brock of England, working independently of each other, corrected the stenotic openings of the pulmonary valve Smithy and his associates worked on the problem of aortic stenosis prior to his untimely death in 1918 In 1950, Bailey continued his previous success in valvular surgery by focusing his attention on the aortic valve He opened the stenosis in the line of the fused commissures and called the operation aortic commissurotomy Hufnagel reported his plastic valvular prosthesis for aortic insufficiency, in 1951

Bailey in Philadelphia, Swan in Denver, Shumacker in Indianapolis Gross in Boston, Dodrill of Detroit, Gordon Murray in Toronto Kirklin in Rochester, Minnesota, Husfeldt, Crafoord, and Bjork from the continent, were reporting their experimental results of the production of atrial septal defects and surgical closure

1955 produced great work by Lillihei in open heart surgery which enabled interventricular septal defects to be successfully closed In Philadelphia, Nichols devised a satisfactory operation for mitral insufficiency in the same year

All great contributors have been thwarted by delay and failures but have repeatedly temporized the remorse of a failure to achieve success by persistent drive and imagination It is axiomatic that each cardiac surgeon has an equally dedicated anesthesiologist It is only by close co operation of the team that acceptable results can be achieved An incomplete list of the groups now working together who have made major contributions in the fields of surgery on the heart and anesthesia for cardiac surgery is included

In 1935 interest in the surgical treatment of coronary artery disease was stimulated by Claude Beck when he published a report on an attempt to revascularize the myocardium

In 1937 John Streider, of Boston, was the first to attack surgically the congenital defect of patent ductus arteriosus this was achieved by ligation but the patient died a few days postoperatively In 1939 Robert Gross also of Boston reported the first successful cure of a patient with a patent ductus arteriosus

As a result of the experiences of World War II anesthesiological techniques and practices were improved and chemotherapeutic agents were inaugurated Dwight Harkin working with his anesthesiologist Charles Burstein reported their success in removing foreign bodies from the heart and great vessels

Crafoord in Europe Gross Blalock and Clagett in the United States were able to correct coarctation of the aorta, and reported independently of each other in 1944

Ingress into cyanotic congenital heart disease was opened in 1945 when Blalock and Taussig reported their work *The Surgical Treatment of Malformations of the Heart in which There is Pulmonary Stenosis or Pulmonary Atresia* The following year Willis Potts of Chicago and his associates amongst them his anesthesiologist William O McQuiston reported their contribution for infants with congenital heart disease

On June 12 1946 Charles P Bailey of Philadelphia digitally opened the anterior and posterior commissures of a calcified mitral valve The operation was named mitral commissurotomy by Thomas Durant of Philadelphia The first successful case was anesthetized by the author for Bailey two years later on June 10 1948 The patient is still alive and symptom free eight years later at the time

THE SELECTION OF THE PATIENT

THE ACCEPTANCE of the medical profession of heart surgery is now established. It is necessary for the cardiac anesthesiologist to be familiar with the essential physical signs, symptoms and laboratory data associated with each of the various cardiac conditions for which surgery can be curative or palliative.

The most satisfactory method of selection and evaluation has evolved as a joint conference attended by the cardiac surgeon (to whom originally, nearly all cardiac patients are referred) the cardiologist and the anesthesiologist. It is obvious that each participant should have examined the patient and be familiar with the symptomatology, physical findings and pertinent laboratory data. If the anesthesiologist assumes his position as a member of the team, he will function preanesthetically during the course of anesthesia and postanesthetically. The position of the anesthesiologist may well be likened to that of the pilot of a ship, the surgeon to the captain and the cardiologist to the navigator, all essential for a safe voyage.

It is unfortunately common for the anesthesiologist to have no part in the selection of the patients for heart surgery. O'Donnell and McDermott have quoted the philosophy of Pender as: "Anesthesiologists must rely on the internist to bring the patient to the operating room at the optimum time and in the optimum condition for surgical intervention. Nothing that we as anesthesiologists can do is designed to improve this condition. The best we can hope for is that our ministrations will not affect the patient adversely." There can be little agreement with such an opinion.

Abbott	Galvin	Emory Univ Georgia
Adams	Eversole	Boston Mass
Bailey	Keown	Philadelphia Pa
Bakst	Pallin	New York NY
Barrett	Wylie	London England
Bigelow	Fairley	Toronto Canada
Blades	Coakley	Washington D C
Brock	Rink and Lucas	London England
Brom	de Zwaan and Boeré	Leiden Holland
Diaz	Gutierrez	Havana Cuba
Doghottu	Ciocatto	Turin Italy
Effler	Hale	Cleveland Ohio
Fell	Heckel	Chicago Illinois
Glen	Artusio	New York, NY
Glover	Schotz	Philadelphia Pa
Gross	Smith	Boston Mass
Harken	Vandam	Boston Mass
Hufnagel	McDermott	Washington D C
Husfeldt	Dam	Copenhagen Denmark
Johnson	Eckenhoff	Philadelphia Pa.
Jones	Ligh	Los Angeles Calif
Julian	Sadove	Chicago Ill.
Kay	Mendelsohn	Cleveland Ohio
Klausen	Jacoby	Columbus Ohio
Kent	Patterson	Pittsburgh Pa
Kirklin	Patrick	Rochester Minn
Lam	Dumke	Detroit Mich
Lillehei	Van Bergen	Minneapolis Minn
Longmire	Dillon	Los Angeles Calif
Merendino	Morris	Seattle Wash
Muller	Eastwood	Charlottesville Va
Murray	Evelyn	Toronto Canada
Neptune	Hand	Boston Mass
Potts	McQuiston	Chicago Ill.
Rumel	Smith	Salt Lake City Utah
Sellers	Sellick and Brown	London England
Strieder	Marcus	Boston Mass
Swan	Virtue	Denver Colo
Sweet	Beecher	Boston Mass
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The anesthesiologist was a physician prior to training in anesthesia and unless he becomes nothing more than a skilled technician, he should act as a physician for the patient's welfare.

The knowledge of the pharmacological responses of patients to all drugs commonly used in anesthesia is one of the most vital portions of our training and knowledge. Thus we should know better than any other specialist which candidates are acceptable to our ministrations of depressing drugs. All anesthetic agents with the exception of oxygen, are depressant.

The anesthesiologist must assume that all patients he anesthetizes will survive. It is unfortunate that this assumption is not always true. In many instances candidates will be brought to surgery who do not have sufficient myocardial reserve to tolerate either the anesthesia or surgical intervention.

One of the difficult phases of preoperative evaluation is the psychological trauma that the patient is undergoing. Many are no more than wards of society as a result of their debilitating disease. The candidate looks forward to surgery as a means of the resumption of the normal duties and activities of life. Time and time again, families will bring undue pressure upon all members of the cardiac team to subject members of their family to surgery and anesthesia. The philosophy of a cardiac anesthesiologist is frequently hindered by his desire to help as many people as is possible. It soon becomes obvious that many people will be studied and surveyed for whom no hope for cure or palliation can be held. It is difficult when one has the final selection to deprive a human being of the possible resumption of a life that is productive. However, it is equally difficult to knowingly administer anesthesia to a patient who has no chance for survival. Yet unless the anesthesiologist assumes a firm

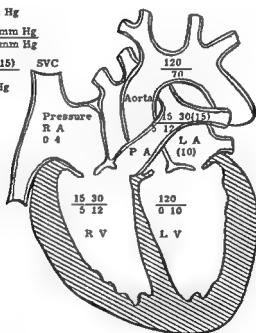
NORMAL HEART
CATHETERIZATION DATA

Pressures Obtained

R A 0.4 mm Hg

R V $\frac{15 \ 30}{0 \ 5}$ mm Hg

P A $\frac{15 \ 30(15)}{5 \ 12}$ mm Hg



L A (10)

L V $\frac{120}{0 \ 10}$

Aorta $\frac{120}{70}$

All Pressures Expressed in mm of Mercury

One becomes rather used to death in this type of work with the mortality rate varying from one disease to another. We are constantly striving to maintain a mortality rate of between 5 and 10 per cent, to be all inclusive for the surgical procedures that are now, routinely, being done. The mortality rates for the various procedures are listed as follows in the succeeding pages.

The congenital heart disease of Tetralogy of Fallot has a mortality rate of 12 per cent. We have had lower operative mortality rates by following the Brock or Sellors procedures in correcting only the pulmonic or infundibular stenosis. Pulmonary stenosis, as an isolated lesion, has a mortality rate of approximately 5 per cent. Atrial septal defects of the ostium secundum type have a mortality rate of approximately 12 per cent; those of the ostium primum variety utilizing closed techniques, have had the highest mortality rate in our series—namely 60 per cent. Atrial septal defects associated with anomalous pulmonary venous drainage, have a death rate of approximately 12 per cent.

Those patients who have patent ductus arteriosus have the lowest of all anesthetic and operative mortality rates for whom anesthesia for cardiac surgery is utilized. The rate should not exceed $2\frac{1}{2}$ to 3 per cent. Willis Potts and William O. McQuiston of Chicago successfully did more than 300 cases without a single fatality. Coarctation of the aorta again has a smaller mortality rate than do the lesions of Tetralogy of Fallot or atrial septal defects. Here the mortality rate ought to be in the neighborhood of 3 per cent. The other congenital lesion, aortic stenosis, has an operated hospital mortality of approximately 7 per cent.

For the acquired heart diseases of valvular origin, mitral stenosis has a death rate of $7\frac{1}{2}$ per cent, and aortic stenosis of 15 per cent. Tricuspid stenosis has never been seen as an isolated lesion, and the mortality connected with this

stand a stigma may be attached to the specialty of cardiac surgery because of an overwhelmingly high mortality rate. We must protect not only the patient but ourselves and all members of the team. Psychological trauma can best be avoided by making careful selection after compiling the physical status of each patient prior to his selection as a candidate for anesthesia and surgery.

The thought that the anesthesiologist is the last person to whom the patient has spoken while alive often plagues us. A very fundamental test of the patient-physician relationship comes as the patient asks the anesthesiologist,

Will I survive this procedure? We have adopted the rule that we tell each patient: There is no such thing as a minor anesthesia. All anesthetics in which the patient loses consciousness are major; the only thing that we can promise is that this is your sole chance for the resumption of normal activities. All of us will do our very best to see that you go through your course safely and in your most physiological state.

Patients in actual heart failure are poor candidates for anesthesia. The higher the patient's degree of physical limitation, the poorer will be the anticipated results. No candidate should be selected if rheumatic fever is present in an acute form. The involvement of more than one valve predisposes to poor results, due principally to the inherent complications of each valvular disease. As the symptomatology is compounded, the satisfactory results are further reduced. In the congenital lesions, particularly in the lower age group, the inherent risk encountered in dealing with children further decreases the satisfactory results of anesthesia and surgery. In children, anesthesia is induced in times of failure of the heart only after we are convinced that medical regimes will produce no better results. However, it is even harder to refuse a child than it is an adult.

PRELIMINARY MEDICATION

THERE CAN BE NO absolute rule written that will cover all the different phases of preliminary medication for heart anesthesia. Time has taught us to be suspicious of over sedation. However, certain generalities can be safely stated as guides for both the congenital lesions and the acquired diseases of the heart.

Each patient is reviewed by the anesthesiologist twenty four hours prior to the induction of anesthesia, and surveyed for preliminary sedation. We consider, individually, the age of the patient, whether the pathology present is acquired or congenital, the duration and severity of symptoms, degree of cardiac reserve, and finally the psychological strain being undergone. The mental outlook is usually excellent in adults. Fear and apprehension are, of course, present but rarely dominant. This single factor of fear is overshadowed by the desire to be benefited by the surgical intervention offered.

During the hospital stay prior to actual scheduling for surgery, free intermingling of preoperative and postoperative patients is encouraged. The cardiologists visit each patient daily and are responsible for the medical preparation. The surgeons and anesthesiologists also visit each patient frequently. This does much to decrease the fear of the patient and a good patient-physician relationship is established. Thus the patient has faith in his management, knows from seeing postoperative patients that the mortality rate is low, and that when brought to the operating room, he will be surrounded by familiar faces. This policy has been immeasurably beneficial in allowing narcotics to be eliminated except in the very young patient (with a con-

disease is the same as for the other associated lesions

Mitral insufficiency at the present time has a death rate of about 12 per cent aortic regurgitation is in the neighborhood of 25 per cent while combined valvular lesions of the acquired type will approximate 20 per cent Coronary artery disease for which surgery is indicated doing a revascularization by the techniques described by Thompson Vineberg Beck or Kralick will result in a hospital death rate of approximately 20 per cent Constrictive pericarditis is a disease with a low mortality rate—approximately 5 per cent Ventricular aneurysms are being done with ever increasing frequency and as the knowledge of the lesion is enhanced the operative mortality is decreased

In the final analysis of the selection of patients last minute cancellation of surgery should always be considered if it is observed by the anesthesiologist that the medical preparation has not been adequate Such cancellations should be decided by mutual agreement between the surgeon and the anesthesiologist each of whom respects the opinion of the other A patient's life may be lost if good advice is not heeded or if the anesthesiologist hesitates to assert himself

testinal complaint following therapeutic doses of morphine

Originally, a barbiturate was ordered for the specificity of protection against procaine reaction as procaine was routinely used in 0.2 per cent solution during anesthesia. Our results have been sufficiently satisfactory since 1948 using pentobarbital (Nembutal) and secobarbital (Seconal) in slight to moderate doses so that we believe some short acting barbiturate ought to be used in all cases unless hypotension or known drug sensitivity exists.

Secobarbital or Nembutal are used interchangeably and are ordered given by mouth ninety minutes prior to the scheduled hour of anesthesia. The blood pressure recording governs the dosage in the following manner: THE BASAL POSITIONAL PRESSURE IS OBTAINED (the palpatory systolic pressure as ascertained with the patient in the same position to be used during the course of anesthesia but prior to receiving any drug that might depress the cardiac output) and recorded. If the blood pressure is less than 90 mm of mercury, no barbiturate is ordered. If the basal positional pressure is between 90 and 100 mm of mercury 50 mg ($\frac{3}{4}$ gr) is ordered. With the few patients where the basal positional pressure is greater than 100 mm of mercury 100 mg ($1\frac{1}{2}$ gr) is ordered.

Thiopental sodium (Pentothal) is administered intramuscularly to infants and children if they are brought to the operating room inadequately sedated. Scott Smith was the first to make us aware that the procedure was safe and relatively free of complications. Pentothal has been used successfully for basal hypnosis in over 300 infants and children at Hahnemann Hospital in Philadelphia, Pennsylvania. We have not had a single case of tissue slough or abscess formation when we have used the intramuscular route. The proper dose is calculated to be 6 to 10 mg per pound of body weight for children less than six years of

genital defect) or the rare, acquired lesion in which pain is a dominant complaint

Adriani has listed five reasons for ordering preanesthetic medication (1) psychic sedation (2) reduction of metabolic rate (3) to obtain an additive effect between a non potent drug and one which is more potent (4) abolition of secretions from the respiratory tract and (5) prophylaxis to avoid anticipated undesirable effects

A barbiturate of short action is ordered for oral ingestion at the hour of sleep the night previous to the induction of anesthesia The dose varies from 50 to 200 mg ($\frac{3}{4}$ gr to 3 gr) depending upon the reflex irritability and complacency of each patient

Meperidine (Demerol) is used in all patients with congenital heart or great vessel pathology The usual dosage ordered is one milligram per pound of body weight—not to exceed 100 mg Demerol is utilized as a premedicant in acquired lesions if pain is present and is of a severe type i.e. coronary artery disease or angina associated with aortic insufficiency Meperidine produces hypotension when administered in large doses or intravenously in patients with heart disease Thus the amount ordered should not be excessive or administered other than by hypodermic injection The time prior to induction of anesthesia ought to be forty five to ninety minutes with one hour being optimal

Barbiturates have proven to be more efficacious than narcotics as tranquilizers for patients with acquired valvular pathology Beecher has stated that less respiratory depression occurs with the usage of barbiturates in achieving a mentally relaxed patient than would accompany an equal effect from narcotics It is certainly true that postanesthetic nausea and vomiting is reduced by the oral administration of secobarbital (Seconal) or pentobarbital (Nembutal) in comparison with the deleterious effect of this gastroin-

ANESTHESIA MANAGEMENT

THE BASIC PRINCIPLES of a properly administered general anesthesia are utilized for patients with cardiovascular abnormalities. These include adequate oxygenation of all tissues, the preservation of adequate cardiac action, and decreasing the irritability of the heart. All factors such as nausea, vomiting, hypotension, hypertension, carbon dioxide excess, or hypoxia must be meticulously avoided.

It is essential that a second by second evaluation of the anesthetized patient be maintained. Individuals with lesions of the cardiovascular system can be compared with tight rope walkers. Life is maintained by a very narrow margin, and a push in either direction may rapidly produce disaster. It is actually preferable that two anesthesiologists be in attendance during the course of anesthesia for heart surgery to complement each other in ascertaining the status of the degree of oxygenation, cardiac output, to control fluid therapy, and to keep adequate meaningful records.

The most serious anesthetic complications encountered during anesthesia for heart surgery are oxygen want, hypotension, cardiac arrhythmias, carbon dioxide retention, anesthetic overdosage, and cardiac asystole.

Oxygen deficiencies are best managed by prevention. To secure this end result, a higher arterial oxygen tension is present during induction and intubation if the patient is receiving oxygen by mask. There is, however, a single factor that keeps us from making this a routine—many cardiac patients have such a fear of suffocation that they

age Hypnosis usually occurs in ten to fifteen minutes Laryngospasm has been noticeably absent, and the major problem has been associated with primary respiratory depression if the amount used exceeds 10 mg per pound of body weight Care must be taken to avoid the sciatic nerve during intramuscular injection

Atropine sulfate has proven to be the belladonna derivative of choice The vagus nerve is inhibited more directly, resulting in a more predictable pulse rate at the time of induction Delirium has largely been avoided and the tracheobronchial tree has been drier

There are two other factors that are used to regulate the preanesthetic dosage of atropine sulfate They are the basal heart rate (optimal compensated ventricular rate without heart failure) and the type of valvular deformity existing in the patient Mitral and tricuspid insufficiencies dictate that the basal heart rate must be eighty per minute or less This is because inadequate filling time for the ventricles precludes adequate ventricular output

Adults with stenotic lesions do better during anesthesia—all other things being equal—with a pulse rate varying between eighty to one hundred per minute Atropine sulfate 0.65 mg (1/100 gr) is ordered if the pulse rate is sixty per minute or less or 0.4 mg (1/150 gr) if the pulse rate is between sixty and eighty per minute if faster than eighty per minute 0.3 mg (1/200 gr) is ordered

Insufficient valves require correspondingly smaller dosages Note well that the basal pulse rate should be slower to allow adequate filling Atropine sulfate should not exceed 0.4 mg (1/150 gr) on any occasion if mitral insufficiency is the prime pathology

Atropine sulfate for children is based on two additional factors—age and size Doses varying from 0.1 mg (1/600 gr) to 0.2 mg (1/300 gr) are usually used and ordered to be given sixty minutes prior to the scheduled time of anesthesia induction

A solution of atropine sulfate should also be selected, a needle introduced through the rubber top of the container, so that if needed it will be within arms reach instantly

A mouth prop is placed on the top of the anesthesia table so that it can be inserted prior to the removal of the laryngoscope from the patient, after intubation

It is relatively essential that the anesthesiologist check to see that the requisitioned citrated blood type is available and that each container of blood lists the patient's name, blood group and type

The anesthesia supplies and equipment should be prepared and checked prior to the patient being brought to the surgical theater. We believe that one reason for the safety record of commercial air lines has been the adherence of the flight crews to the rules for checking and rechecking their equipment prior to being airborne, and that the same is true for cardiac anesthesiologists

All of this preparation requires time but short cuts are prime factors in the precipitation of catastrophe, and no place is this more true than in attempting anesthesia for heart surgery, without adequate expenditure of the time necessary for comprehensive preparation

The operating room personnel should not conduct loud conversations and should be especially trained to be quietly efficient. Very few stimuli are comparable to a dropped basin full of surgical instruments to upset a patient lying on an operating room table awaiting heart surgery

As soon as the patient has been brought to the operating room and transferred from the carrier to the operating table the anesthesiologist should make himself known to the patient. A kind word does much to ease the mental tension. The blood pressure cuff and stethoscope should be applied quickly and the blood pressure and pulse rate recorded on the anesthesia chart. All cannulae for infusions

manifest major annoyance at having a mask placed over their faces. Rather than produce undue strain or apprehension by insisting that a mask be secured in place during induction in these lightly premedicated patients oxygen is utilized only when the patient does not object. We recognize the theoretical and practical values but are equally familiar with the patient's objections.

We have a check list for equipment and supplies that are used or may be used during the anesthetic period and each item is prepared and placed in a readily available location before induction. The list includes a laryngoscope with a blade of proper size that functions perfectly because a light bulb that fails to function during intubation is not conducive to a well managed operative course. Three assorted endotracheal catheters with inflatable cuffs are selected the adjudged proper size and one each the next size larger and smaller. In each instance the cuff is inflated and placed under water to detect leaks. The carbon dioxide absorber is filled and a spare cannister is provided for rapid interchange. The oxygen supply is rechecked and a supply of at least two extra tanks provided so that at no time will we be without an adequate reserve supply during anesthesia. The nitrous oxide, cyclopropane and ether supplies are ascertained.

We daily prepare fresh solutions of thiopental sodium 40 mg of methoxamine hydrochloride (Vasoxyl) in 500 ml of 5 per cent glucose and distilled water. 4 ml of 0.2 per cent (8 mg) levarterenol bitartrate (Levophed) is prepared by diluting it in 500 ml of 5 per cent glucose and distilled water. a third vasopressor solution is prepared by adding 40 mg of methoxamine hydrochloride (Vasoxyl) and 2 ml of levarterenol bitartrate (4 mg) to a solution of 5 per cent glucose and distilled water. 3 ml of 1/1000 (3 mg) epinephrine chloride is added to 500 ml of 10 per cent glucose and distilled water.

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should be rechecked to ascertain patency prior to actual induction of anesthesia

There is an iron-clad rule in our operating rooms that no induction of anesthesia will be permitted until the suture nurse is scrubbed gowned and gloved and until she has checked her instruments and set her table. There must be at least one responsible surgeon in the operating room scrubbed gowned gloved and ready to operate. This is not a whim of the anesthesiologist but a rule enforced because of his knowledge based on experience that regardless of how carefully the patient has been prepared and selected cardiac arrest may occur during induction. When such a complication is produced by anesthesia time is of the utmost importance and can not be wasted looking for a surgeon to institute thoracotomy and manual compression of the heart. Obviously surgical instruments are necessary—hence the requirements placed on the nursing and surgical staff.

Anesthesia is induced by injecting a muscle relaxant through a three way stop-cock into a vein. We prefer dexamethonium bromide (Syncurine) as the relaxant for endotracheal intubation. Thiopental sodium (Pentothal Sodium) is next administered intravenously and the trachea is intubated under direct vision laryngoscopy using the oral route. We use the largest bore rubber catheter with inflatable cuff that can be introduced without producing trauma. The mouth prop is inserted the laryngoscope removed from the oral cavity and the patient's lungs inflated with oxygen by manual compression of the breathing bag. An assistant should palpate a peripheral artery (radial) from the start of anesthesia until the completion of intubation at that time the systemic blood pressure should be ascertained.

The patient is then positioned for surgery the pulse rate and the blood pressure are again recorded. If the pressure

is less than preanesthetically, only oxygen is administered. If hypotension (palpatory systolic pressure of 80 mm of mercury) exists, the case should be cancelled and the patient awakened as quickly as possible. Nitrous oxide in equal amounts with oxygen is utilized if the systolic systemic pressure is 100 mm of mercury or more.

Respirations are controlled from the time of intubation until the pleura has been closed and a pneumothorax no longer exists. The rate of the respiratory cycle may vary from twenty to thirty per minute without ill effects. The inspiratory phase is positive and the expiratory phase should have a zero pressure. We prefer a one to one cycle with equal times of inspiration and expiration. Five liters of inspired gas per minute has proven satisfactory in all of our adult cases. The carbon dioxide absorber is always in the circuit, and with a semi-closed technique, we believe carbon dioxide retention to be abolished. Carbon dioxide is a waste product of respiration and excess predisposes to cardiac dilatation.

Thiopental sodium is added as needed to produce hypnosis and facilitate the positive pressure inspiratory period of respiration. Individuals with profuse secretions of the tracheal bronchial tree demand either overdosage with thiopental sodium or, preferably the use of a muscle relaxant such as succinylcholine hydrochloride during aspiration through the endotracheal catheter.

Citrated whole blood is used as needed. An aspiration container that traps blood loss will give a rough guide as to the degree of hemorrhage. In general, during anesthesia for cardiac surgery it is better to undertransfuse than to overtransfuse.

There are many advocates of ether analgesia. Others prefer cyclopropane and ether, while still others give glowing reports about various other techniques. Each anesthesiologist should use the agents and techniques with which he is

most competent but keep an open mind about the limitations of all agents. The essentials are adequate oxygenation, constant evaluation, and a light plane of anesthesia. The plan of the outline above was reached after careful observation and a fair trial with other recommended agents. It affords a technique that assures adequate oxygenation, in-take, minimal depression of cardiac reserve, and uses drugs that have short duration of action.

We used a solution of 0.2 per cent procaine hydrochloride in 1800 consecutive cases. Procaine hydrochloride has several distinct advantages: it reduces the total amount of required intravenous barbiturates by one third; the pulse rate can be more readily controlled, and we believe it affords some analgesia. The disadvantages in the use of procaine hydrochloride are an absolute contraindication when the patient has been prepared with quinidine sulfate, as cardiac asystole is more prone to occur; hypotension can be aggravated; and a false sense of security results as to the prevention of cardiac arrhythmias. It can categorically be stated that procaine will not preclude ventricular tachycardia or ventricular fibrillation.

We use electrocardiography routinely for only three types of cases: (1) whenever hypothermia is used as an adjunct so that occult shivering can be detected; (2) during the repair of interauricular septal defects so that it is possible to detect an interruption of the auriculoventricular conduction system; and (3) whenever extracorporeal circulation is indicated.

An anesthesiologist should be able to detect conduction abnormalities or cardiac arrhythmias by careful palpation of a peripheral artery and by direct vision of the heart.

A general classification of heart rate abnormalities is included.

CARDIAC IRREGULARITIES WITH WHICH THE ANESTHESIOLOGIST SHOULD BE FAMILIAR

GENERAL CLASSIFICATION

- A. Arising in Sino-Auricular node
 - 1 Sinus arrhythmia
 - 2 Sinus bradycardia
 - 3 S.A. Heart Block
 - 4 Wandering pacemaker
 - 5 Sinus tachycardia
- B Arrhythmias from Auricles
 - 1 Auricular premature systoles
 - 2 Auricular paroxysmal tachycardia
 - 3 Auricular flutter
 - 4 Auricular fibrillation
- C Arising in Auricular Ventricular node
 - 1 A V Block
 - 2 A V nodal rhythm
 - 3 Nodal premature systoles
- D Disturbances of Ventricles
 - 1 Ventricular premature systoles
 - 2 Ventricular tachycardia
 - 3 Ventricular flutter
 - 4 Pulsus alternans
 - 5 Ventricular fibrillation

SINUS ARRHYTHMIA

Sinus arrhythmia is known to be present when the sinus node undergoes cyclic variations of rate

Sinus arrhythmia is seen mostly prior to the age of twenty one and in the very elderly patient

In the operating room sinus arrhythmia frequently is confused with auricular fibrillation. If the patient is asked to hold his breath while you listen to his heart, then is

asked to breathe deeply, the sinus arrhythmia will disappear. During anesthesia, if either atropine sulfate or general anesthesia is used, this irregularity is seldom if ever seen.

Sinus arrhythmia is the most frequent of all arrhythmias found in the resting human free of disease. Ordinarily no medication is needed or indicated.

SINUS BRACHYCARDIA

Sinus Brachycardia exists when the heart rate is below sixty per minute, and no conduction block exists.

Acute episodes of sinus brachycardia are a result of vagal stimulation. It is found in increased intracranial pressure, increased cerebrospinal fluid pressure, gastric dilation, biliary or renal colic.

Atropine sulfate is the drug of choice.

S A HEART BLOCK

S A heart block clinically can not be differentiated from atrial dropped beats. Fortunately sino atrial block has little clinical significance and there is no therapy needed except for associated heart disease if any is present.

WANDERING PACEMAKER

Wandering pacemaker can only be diagnosed by ECG and there is usually no disturbance of rhythm.

SINUS TACHYCARDIA

Sinus tachycardia is present whenever the heart rate exceeds 100 per minute. It is possible for the rate to be as rapid as 240 per minute. Generally the rate varies from 120 to 140 per minute. The rhythm is regular and produced most frequently by fear, worry, fever, nicotine, alcohol and atropine sulfate.

AURICULAR PREMATURE SYSTOLES

Arrhythmias from the auricles may arise in a portion of the sinus node or in any part of the atrial tissue

Auricular extra systoles can be diagnosed by careful palpation of the pulse, as atrial premature systoles are not followed by a full compensatory pause as are ventricular premature systoles

This inequality is found in patients with severe infectious disease and during the course of anesthesia, as well as following coronary artery disease. They rarely occur in normal hearts unless fever causes a disturbance of the metabolism of the atrium

AURICULAR PAROXYSMAL TACHYCARDIA

Auricular paroxysmal tachycardia usually begins and ends abruptly. The rate is perfectly regular and may vary from 100 to 250 per minute. Peripheral vascular collapse is not uncommon due to poor filling time and a backward failure ensues

Thirty four per cent of patients with auricular paroxysmal tachycardia have normal hearts, the remaining portion equally divided between rheumatic heart disease and arteriosclerosis

The diagnosis is made by suddenness of onset, rapid rate and response to vagal stimulation such as eyeball pressure, carotid sinus pressure or induced vomiting. If the rate remains rapid following vagal stimulation an ECG should be taken for positive diagnosis and prior to drug therapy

AURICULAR FLUTTER

Auricular flutter may be considered a more serious form of atrial tachycardia, the main difference being the rate and site of origin of the ectopic focus. The rate of the auricle may increase to 400 per minute. The ectopic focus is low—

near the stoma of the inferior vena cava. There is usually a 2:1 or 3:1 block. The radial pulse is then about the same in atrial flutter as it is in atrial tachycardia.

Since atrial flutter is caused by an ectopic focus far from the sino auricular node it is not influenced by vagal stimulation as is atrial tachycardia.

Atrial flutter should be regarded as evidence of organic heart disease and therefore treatment is indicated. Quinidine affords excellent results if the heart is not decompensated. If failure is present digitalis is preferred. This has the distinct advantage in controlling the ventricular rate and decompensation is relieved. ECG shows saw tooth P waves.

AURICULAR FIBRILLATION

Auricular fibrillation: the rate of activity of the auricle is seldom less than 350 per minute. Rates of 500-600 have been described from the ordinary ECG. The ECG tracings reveal no P waves and no two QRS complexes are the same interval apart. Clinically the diagnosis is made by a pulse deficit between the apex and the radial pulse and the irregularity of the pulse. The auricles lose all characteristics of systole and serve only as a reservoir for blood; this is followed by atrial dilation with a resultant decrease in cardiac output.

Therapy is dependent upon the degree of myocardial damage and the benefit of reverting the rhythm to a normal sinus rhythm. Digitalis and quinidine are the two drugs most frequently used to control atrial fibrillation. Quinidine is the only drug capable of producing a reversion to normal rhythm.

A.V. BLOCK

A V block is the failure of impulses arising in the auricle to be transmitted directly to the ventricle.

For the sake of simplicity, A V blocks are classified as

1 First degree, which is a prolongation of the conduction time beyond normal

2 Second degree varies from a block which permits only every second atrial impulse to pass (2 1) to one which permits only a few of the atrial impulses to pass (1 1)

3 Third degree (complete A V block) all of the atrial impulses fail to reach the ventricle Two pacemakers in the heart, one in the atrium and one in the ventricle

The most frequent cause of A V block is old age Heart muscle may have depressed excitability but still appear normal histologically For this very reason even with severe heart block being the cause of death, no microscopic findings can be found to substantiate the diagnosis

Poisons drugs and fever all produce block Digitalis and quinidine are the most frequently used drugs clinically capable of producing heart block Prolonged fever of any nature, including the fever produced by physical exercise exhausts the heart and depresses excitability

Generally heart block does not exist in the absence of organic heart disease

Atropine sulfate has little clinical effect, a sympathomimetic drug (ephedrine sulfate or amphetamine) and oxygen render the best results, particularly if the pulse rate is thirty five to forty per minute

A V NODAL RHYTHM

A V nodal rhythm is present when the A V node acts as the pacemaker Under this condition the impulses originate in the A V node and spread from there to the auricles and to the ventricles Clinically it is undetectable with the ventricles and the auricles contracting synchronously Usually the rate is from fifty to sixty per minute The clinical application is as a precursor to atrial tachycardia flutter or fibrillation

NODAL PREMATURE SYSTOLES

Nodal extrasystoles may occasionally arise at the A.V node This can not be diagnosed clinically by auscultation or arterial palpation

VENTRICULAR PREMATURE SYSTOLES

Ventricular extrasystoles are the most common of all arrhythmias By auscultation premature beats are detected by their prematurity and the compensatory pause Ventricular extrasystoles occur in every person they can be due to fatigue or actual cardiac disease

When premature ventricular systoles occur during general anesthesia the oxygen concentration should immediately be increased. If the arrhythmia continues small amounts of barbiturates will ordinarily abolish the irregularity except in the presence of coronary artery disease

VENTRICULAR TACHYCARDIA

Ventricular tachycardia when sinus rhythm becomes superseded by a ventricular ectopic focus of rapid rate the resulting arrhythmia is called ventricular tachycardia The rate tends to be in the neighborhood of 200 per minute

The presence of a rapid thready pulse and distant heart sounds with hypotension and a narrow pulse pressure in patients known to have cardiac pathology is most indicative of ventricular tachycardia Vagal stimulation does not decrease the heart rate and so it may thus be differentiated from atrial tachycardia

Therapy is indicated immediately and quinidine and quinidine like drugs are most commonly used DIGITALIS IS CONTRAINDICATED because it may produce ventricular fibrillation Oxygen and Methoxamine hydrochloride have proven very useful in the operating room to control this irregularity slowing the pulse rate

elevating the cardiac output, and increasing the systolic pressure

VENTRICULAR FLUTTER

Ventricular flutter is an ECG variant of ventricular tachycardia. Clinically there is no difference between the two. The treatment is the same.

PULSUS ALTERNANS

Pulsus alternans produces alternately strong and weak radial pulsations. There are corresponding loud and distant heart sounds at the apex (there is no variation of the ECG complexes). It may occur during myocardial infarction, with left ventricular failure or anoxia.

During blood pressure determinations by the auscultatory method two systolic levels vary from 5 to 15 mm of mercury.

VENTRICULAR FIBRILLATION

Ventricular fibrillation is the most grave of all cardiac irregularities. The only two ways by which the diagnosis can be made are ECG or direct visualization of the ventricles. Clinically it is difficult to differentiate from cardiac asystole.

The treatment is variable with no drug capable of arresting the irregularity once present. Oxygen and artificial respiration are mandatory. Manual cardiac compression is indicated to nourish the ventricles via the coronary arteries. Electric countershock should be employed following adequate oxygenation if manual compression of the heart and epinephrine do not revert the rhythm. Untreated it is 100 per cent fatal.

COMPLICATIONS DURING ANESTHESIA

COMPLICATIONS during anesthesia may arise from several sources anesthesia per se surgical complications patient's disease or errors of judgment Whatever the etiological factor the best therapy is avoidance or removal of the cause Experience is said to be the best teacher and as one who is a firm believer in this philosophy it is possible to write as an authority—all known anesthetic complications having occurred during the course of my career

The hindrances to adequate circulation are those most frequently encountered during heart surgery These include hypotension hypertension arrhythmia heart block primary cardiac failure pulmonary edema and cardiac asystole

Respiratory difficulties that are manifest during anesthesia for cardiac surgery are hypoxia from obstructed air ways inadequate oxygen source preexisting pathological pulmonary changes pleural effusion secretions within the tracheobronchial tree and inadvertent extubation

Hypotension is considered present if the systemic systolic blood pressure is 100 mm. of mercury or less Hypotension is by far the most frequently encountered complication during the course of anesthesia Most often hypotension is simply a manifestation of poor myocardial tone and relatively ineffectual ventricular output It is an obvious truism that the less cardiac impairment preoperatively the more stable is the heart action during anesthesia This is one of the reasons cardiovascular surgeons cardiologists and anesthesiologists make such a strong plea for early surgery

It is, at best, the difference between a cure and a palliation. The existence of a heart murmur is not the indication for surgery, but increasing disability and progression of disease are.

It is better to overcome hypotension on a physiological basis rather than by pharmacological means. This is true when pressure is made on the heart, or against the inflow tract. Simply by standing and observing the position of a retractor, gauze sponge, or surgical assistant's hand, it is possible to check this cause. If the offending pressure can be removed and traction released the systemic pressure will rapidly return to its normal level.

Blood loss is another common factor in the production of hypotension. There is, at present, no accurate way available during surgery and anesthesia, to obtain immediate blood volume determinations. Rough guides are available by means of hematocrit, hemoglobin and erythrocyte determinations. Sponges may be weighed and suction bottles measured, but still this is by no means accurate. We have advocated that the anesthesiologist have an alarm system or a dinner bell and ring it each time a unit of blood is administered. These methods could prove disturbing, but would allow the team as a whole, to be made familiar with the volume of blood used.

There are several vasopressors that are recommended to maintain TEMPORARILY adequate circulation. Much has been written about these agents. We have found three to be useful during anesthesia for cardiac surgery: (1) methoxamine hydrochloride (Vasoxyl), (2) levarterenol bitartrate (Levophed), and (3) epinephrine. Methoxamine hydrochloride is indicated if the pulse rate is rapid and hypotension is present. Levarterenol is used if the pulse rate is slow and hypotension exists. 3 ml of 1 to 1000 epinephrine hydrochloride is added to a 500 ml solution of 10 per cent glucose and water and administered intrave-

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corrected by the use of epinephrine in conjunction with manual compression of the heart and adequate oxygenation of the lungs. It is less helpful in valvular lesions that are regurgitant than in those resulting from stenosis, whether the lesion is congenital or acquired.

Cardiac asystole is, of course, the most alarming complication encountered. It can occur at any time during anesthesia. There are several rather constant etiological factors in its production: (1) oxygen deprivation, (2) anesthetic overdosage, (3) hemorrhage, (4) coronary embolization, (5) administration of improper drugs, (6) adrenal failure, (7) too constant a pressure on the heart, (8) occlusion of a valve orifice by thrombus, instrument or finger, (9) myocardial failure, and (10) carbon dioxide excess. The anesthesiologist must constantly seek to avoid the occurrence of cardiac asystole and can do so to a great degree by obtaining an adequate history from the patient, by constant observation during surgery and anesthesia, and by informing the surgeon promptly of difficulties that predispose to death during surgery.

Hypoxia as a major cause of complications during anesthesia can probably never be overstressed. The body has no reservoir of oxygen or means of producing oxygen during periods of stress when an abundance is so acutely needed. As has been mentioned previously, no single thing is so vital to sustain life or prevent cardiac irregularities. It might be well to recall the old saying that noisy respirations during anesthesia are pathognomonic of obstruction, but that obstructed respirations need not be noisy, i.e., complete.

Oxygen deprivation can largely be avoided by meticulous attention to details. We believe the inspired concentration should never be less than a 2:1 ratio, and most often a 1:1 ratio is desired. Over 50 per cent of all heart cases done in our hospital receive a 100 per cent inspired con-

nously if no vasopressor response is elicited by either of the two previously named drugs. We are fully acquainted with the standard pharmacological textbooks and the warning they contain about whipping a tired horse. We are discussing diseased hearts that develop poor output and there is no tired horse as tired as a dead heart. Epinephrine is a natural stimulant to the heart and as such should be used in times of stress.

Hypertension is a systemic systolic blood pressure of 150 mm. of mercury or greater and/or a diastolic blood pressure of at least 100 mm. of mercury. Hypertension occurs normally in patients with coarctation of the aorta, aortic insufficiency or coronary artery disease.

If hypertension develops during the course of anesthesia for cardiac surgery we consider one of four things to be in effect: (1) hypoxia, (2) carbon dioxide accumulation, (3) the patient is feeling pain or (4) a vasopressor is being inadvertently administered. Proper steps are taken to find the cause and correct the discrepancy immediately.

Cardiac irregularities are discussed in the section on management. However the serious arrhythmias are ventricular tachycardia and ventricular fibrillation. We prefer to treat ventricular tachycardia by injecting a solution of 500 ml. of 5 per cent glucose and water with 40 mg. of methoxamine hydrochloride added intravenously at whatever rate needed to slow the pulse. This will almost invariably slow the pulse rate and elevate the systemic blood pressure. Sodium molar lactate has recently been advocated by Bellett as a useful therapeutic and prophylactic measure. We have used sodium molar lactate eight times with varying degrees of success. Epinephrine has been used to treat ventricular fibrillation in our clinic for several years. It might seem heresy to write advocating epinephrine for the treatment of an arrhythmia that it supposedly causes each time it is used but ventricular fibrillation can be

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POSTANESTHETIC MANAGEMENT

THE DUTIES of an anesthesiologist are not over immediately following the completion of surgery, with bronchial tracheal aspiration and extubation

The immediate postanesthetic period (twenty four hours) is a most critical time for the patient. He has been subjected to the trauma of surgery (no matter how efficiently and quickly accomplished) and the added depression of a general anesthesia. Each patient in the postoperative period has to adjust to a change in hemodynamics. This varies according to the surgery and whether or not the intervention was for obstruction, i.e., any stenotic valve, or coarctation of the aorta, or to changes in intracardiac chambers following the correction of an insufficiency.

Immediately following extubation, oxygen is supplied to the adult patient by means of a B L B mask, with a flow rate of ten liters per minute. The patient should be examined immediately for any evidence of embolization. Cerebral embolization can be detected by inability to speak or loss of motor functions of the extremities. The anesthesiologist should command the patient to move a designated limb if sufficiently conscious or recovered from the effects of general anesthesia. We attempt to have all patients awake at the completion of surgery so that if a cerebral vascular accident has occurred it will be known to us. Peripheral arterial pulsations should be noted, and if found to be absent post surgically having been present pre surgically an embolectomy should be done promptly. We prefer to use either spinal anesthesia or epidural block as the anesthesia of choice for embolectomies, feeling that the sympathetic

centration of oxygen

We have devised a polyvinyl plastic mouth prop through which an endotracheal catheter can be easily inserted. This prohibits occlusion by the teeth. The cannula has a pharyngeal flange that is easily secured in place against the lips with adhesive tape. The endotracheal tube can neither slip further into the trachea nor be pulled out as long as the initial positioning has been correct.

All endotracheal catheters should be measured prior to insertion to avoid inadvertent endobronchial intubation. The endotracheal catheter should be introduced under direct vision laryngoscopy. This provides further protection against kinking of the tube or esophageal intubation.

An aspirating catheter is routinely lubricated and inserted through the endotracheal tube prior to the start of surgery. If the aspirating catheter can not easily be inserted, some evidence of obstruction exists that must be corrected before a pneumothorax is present.

Secretions must be removed and not allowed to accumulate or not only will hypoxia result, but greater depths of anesthesia will be required to combat bucking by the patient.

Pulmonary edema as a complicating factor rarely occurs but when it does develop, emergency treatment is mandatory. The routine for therapy that we use in combatting pulmonary edema is discussed in the chapter on postanesthetic management.

Blood loss from hemorrhage is an excellent example of the needed teamwork for an outcome that will be successful for the patient. Anesthesiologists seldom produce a massive hemorrhage but we should assume the responsibility for adequate and rapid replacement of the depleted circulating blood volume.

Complications may be likened to accidents in that they are always caused and never just occur.

POSTANESTHETIC MANAGEMENT

THE DUTIES of an anesthesiologist are not over immediately following the completion of surgery, with bronchial tracheal aspiration and extubation

The immediate postanesthetic period (twenty four hours) is a most critical time for the patient. He has been subjected to the trauma of surgery (no matter how efficiently and quickly accomplished) and the added depression of a general anesthesia. Each patient in the postoperative period has to adjust to a change in hemodynamics. This varies according to the surgery and whether or not the intervention was for obstruction, i e., any stenotic valve, or coarctation of the aorta or to changes in intracardiac chambers, following the correction of an insufficiency.

Immediately following extubation, oxygen is supplied to the adult patient by means of a B. L. B. mask, with a flow rate of ten liters per minute. The patient should be examined immediately for any evidence of embolization. Cerebral embolization can be detected by inability to speak or loss of motor functions of the extremities. The anesthesiologist should command the patient to move a designated limb if sufficiently conscious or recovered from the effects of general anesthesia. We attempt to have all patients awake at the completion of surgery so that if a cerebral vascular accident has occurred it will be known to us. Peripheral arterial pulsations should be noted and if found to be absent post surgically having been present pre surgically, an embolectomy should be done promptly. We prefer to use either spinal anesthesia or epidural block as the anesthesia of choice for embolectomies feeling that the sympathetic

nerve inhibition overcomes spasm in the arteries and aids the collateral circulation. The comparison of preanesthetic and postanesthetic oscillometric recordings in the lower extremities has proven most helpful in taking the guess work out of ascertaining the actual existence of an adequate blood supply.

The anesthesiologist should ascertain whether or not the lungs are completely inflated while the patient is still on the operating room table. This can be accomplished in several ways: auscultation of the breath sounds and by determining the negative pressure present in the underwater seal bottle. These are the simplest methods available. A routine chest plate can be made, but this seems expensive, troublesome and unnecessary to us.

We insist that oxygen be administered by mask until the patient is thoroughly conscious and the systemic blood pressure is stabilized at a satisfactory level. Following the administration of oxygen by a mask, we use nasal catheters to administer oxygen. The oxygen should be humidified by bubbling through water and the rate should not exceed five liters per minute. The nasal catheter should be changed every eight hours; a clean catheter introduced into the opposite nares will do much to provide comfort to the patient and make him more prone to accept the inconvenience of oxygen therapy.

It may be well to recall that there are three generally used methods of administering oxygen: (1) by mask, (2) by nasal catheter, or (3) in a tent. Each of the three modes of inhalation therapy has advantages and disadvantages. The usual concentration of oxygen in the inspired air by mask with a flow rate of ten liters per minute is 95 to 100 per cent. Nasal oxygen allows a concentration of oxygen inspired with air of 35 to 40 per cent at five liters per minute. The concentration of oxygen inspired under a tent is 35 to 50

per cent dependent upon the flow rate and the lack of leaks in the canopy

Postanesthetic sedation should be ordered by the anesthesiologist for the first twelve to twenty four hours. We prefer to administer no narcotics until after the patient is fully awake, responsive, and has a satisfactory circulatory system. There can be no question but that adequate nursing care markedly curtails the amount of sedation needed following surgery. Although talking and reasoning do not replace adequate control of pain, an attentive nurse can do much to allay apprehension and so decrease the sedation schedule. It is necessary that all post thoracotomy patients cough and expectorate secretions thus minimizing the amount of retained secretions and avoiding to a large degree the development of atelectasis.

Hypotension is a rather common postanesthetic problem. It should be recalled that hypotension is best treated physiologically by removing the cause. A decrease in cardiac output and low blood pressure, however, must be corrected rapidly no matter what the cause. If untreated, renal failure will ensue and death will result from uremia. Of course, it must be borne in mind, that there is an abnormal renal hydrostatic pressure in nearly all patients with acquired heart disease, and renal complications are more prone to develop following the further depression attendant to hypotension. In our research laboratory and in clinical practice levarterenol (Levophed) produced less constriction of the renal arterioles than the other commonly used vasopressors. The prolonged use of levarterenol should not be considered as untoward effects result such as tissue slough and gangrene. Certainly a judicious survey should be made as to the existing blood volume in the immediate postanesthetic phase. This will mean hematocrit erythrocyte hemoglobin and if possible blood volume determinations. The

use of citrated whole blood is a life saving procedure when indicated but unfortunately, the use of blood transfusions is not a completely benign process. It is easy to overload a heart with the rapid administration of blood and produce pulmonary edema. Also transfusion reactions do occur, although with an ever-decreasing frequency.

Pulmonary edema may occur from any one of several causes in the postanesthetic period. As mentioned above, it may follow the rapid administration of citrated whole blood or too large a volume of blood may be given to a laboring heart precipitating pulmonary edema. Hypoxia, regardless of the cause—be it respiratory obstruction, respiratory failure from irreversible lung changes, primary depression of the respiratory center from overdosage with a narcotic or inadequate oxygen concentration in the inspired atmosphere—is a major factor in the development of pulmonary edema. Cardiac failure per se may well be the major etiological factor. Whatever the cause the treatment should be immediate and effective or death will rapidly result. Perhaps this is one phase of medicine that should be routinized into a cookbook fashion and followed closely by all physicians dealing with pulmonary edema in the post surgical period of patients who have just had heart surgery.

The clinical picture is usually typical. A sudden onset of dyspnea and cyanosis with signs of circulatory collapse are seen first. White frothy sputum appears and soon becomes pink in color indicating bloody content. The secretions may be profuse and literally drown the patient in a matter of a few minutes. Crepitant rales may be heard over the entire lung field.

The first step is to provide adequate oxygen to the patient. If still in the operating room, this is best achieved by means of an anesthesia machine. The oxygen should be ad-

ministered under positive pressure. We use as much as 40 mm of mercury during inspiration. The expiratory phase should probably be maintained on the positive side, so that a negative pressure does not exist in the respiratory cycle. If the patient is on the ward or in his room, any positive pressure mask may be used with good results unless apnea ensues, in such a case, a means of affording respirations must be obtained. Various anti foam agents have been used. Certainly one is indicated and we prefer caprylic alcohol one part mixed with six parts water.

A bloodless phlebotomy should be immediately instituted. This may be achieved in one of two ways i.e., by applying tourniquets to three extremities or by actual administration of spinal anesthesia to a level of the tenth thoracic dermatome. The drugs that are useful are Lanatoside C 1.2 to 1.6 mg and Aminophyllin 0.25 to 0.5 grams intravenously. The tracheobronchial tree should be aspirated frequently but not at the expense of decreasing the inspiration of oxygen and the defoaming agent. We have no place for morphine sulfate or any of the other narcotics in the therapy of pulmonary edema in the immediate postanesthetic period. We mention their wide spread use only to condemn it.

Actual phlebotomy is indicated if there is not a pronounced decrease in the rate of production of the pulmonary edema in ten to fifteen minutes. The blood should be removed slowly from a vein. The phlebotomy should be discontinued as the production of edema is evident. Shock from blood loss could easily cloud the clinical field if carried to extremes.

Hypothermia is utilized by slow deliberate means if patients develop fever above 102° Fahrenheit in the post anesthetic period. We prefer a water cooled mattress, as this has three distinct advantages. (1) it can easily be in

stalled in the patient's room, (2) is more comfortable for the patient and (3) the speed of cooling (reduction of body temperature) is controllable. Each elevation of body temperature of 1° Fahrenheit increases the basal metabolic rate by 7 per cent. It is a well established fact that fever increases the pulse rate. Temperature recordings less than normal reduce the oxygen demand of the myocardium and conserve body energies.

The anesthesiologist should visit the patients after surgery to continue the physician-patient relationship. We should always recall that physicians are trained to aid patients not patients to aid physicians.

TETRALOGY OF FALLOT

FALLOT, in 1888, published his classic treatise on *The Anatomic Pathology of the Cyanotic Heart*. Four congenital pathological conditions were listed: (1) pulmonary stenosis or pulmonary atresia, (2) right ventricular hypertrophy, (3) interventricular septal defect, and (4) dextro-position of the aorta.

The course of the circulation in patients with the tetralogy is best described by starting with the blood in the right atrium, passing into the right ventricle through the tricuspid valve. A variable portion of the venous blood enters the pulmonary artery during the systole of the right ventricle. This portion will then undergo normal oxygenation via the pulmonary circulation. The major volume of blood, due to the obstruction of the pulmonary artery, is shunted from the right ventricle, via the interventricular septal defect to the left ventricle. The mixing of the oxygenated and unoxygenated blood occurs in the left ventricle and aorta. Left ventricular systole forces the mixed blood into the aorta and out into the systemic circulation.

Symptoms vary according to the severity of the obstruction to the pulmonary artery. The obstruction may be in fundibular, valvular or due to a primary pulmonary artery atresia. Slight exertion produces an increase in the degree of cyanosis. It may be well to recall that at least 5 grams of reduced hemoglobin per 100 ml of blood are necessary to manifest cyanosis. Dyspnea and fatigue are the prime symptoms, frequently being precipitated by crying or eating. Periods of unconsciousness result from oxygen deprivation.

vation to the brain Cerebral thrombosis with resultant paralysis may occur

Physical diagnosis can never be 100 per cent correct without laboratory confirmation Typically, the child is retarded physically but not of necessity mentally sluggish Cyanosis of the lips, tongue conjunctiva and nail beds is present The digits are clubbed There is a harsh systolic murmur heard over the pulmonic valve although the degree of obstruction to the pulmonary artery produces marked variations in the intensity of the murmur

The child assumes a characteristic squatting position after exertion of even a moderate degree Infants unable or too young to walk flex their thighs against the abdomen in a similar manner Generally the heart size is normal or only slightly enlarged

Several laboratory studies are of value in diagnosis and in evaluation of the severity of the lesion Polycythemia is the rule, red counts of seven or eight million being not uncommon

On x ray, the pulmonary vascular markings are diminutive In most patients the main pulmonary artery cannot be defined The heart is as a rule not enlarged The left border is rounded and the apex elevated In the right anterior oblique view there may be anterior prominence of the right ventricle In the left anterior oblique view the aorta frequently is more anterior than normal

The electrocardiogram almost invariably shows right ventricular hypertrophy

Angiocardiography demonstrates simultaneous opacification of the right ventricle aorta and a small pulmonary artery Using high speed techniques the location of the pulmonary obstruction may be outlined

On cardiac catheterization there is found to be a gradient in systolic pressure from the pulmonary artery to the

TETRALOGY OF FALLOT

CATHETERIZATION DATA

Pressures Obtained

$$R \text{ V } \frac{95}{0}$$

$$R \text{ P A } \frac{20(13)}{10}$$

$$B \text{ A } \frac{98(84)}{70}$$

$$L \text{ A } (0)$$

$$M \text{ P A } \frac{16(11)}{5}$$

$$(0) \text{ mm Hg}$$

$$R \text{ A.}$$

$$\frac{95}{0}$$

$$\text{mm Hg}$$

$$R \text{ V}$$

Normal Values

$$B \text{ A. } 0.4 \text{ mm}$$

$$R \text{ V } \frac{15 \text{ } 30}{0 \text{ } 5}$$

$$P \text{ A } \frac{15 \text{ } 30(12)}{5 \text{ } 12}$$

All Pressures Expressed in mm of Mercury

Oxygen Values Expressed in Volumes per cent

O₂ Consumption 130 cc/min

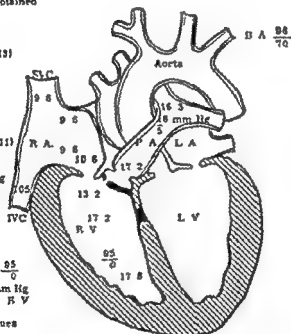
Pulmonic Blood Flow 2.4 l/min

Systemic Blood Flow 3.3 l/min

Shunts L to R 1.4 l/min

R to L 2.3 l/min

Overall Shunt (R to L) 0.9 l/min



BA
Cont 14.0
Cap 33.4
Sat 80%

right ventricle. Frequently the catheter tip can be manipulated through the ventricular septal defect into the aorta. The systolic pressure in the right ventricle and the systemic circuit is as a rule, equal. There may, or may not be, an increase in the oxygen content in the outflow of the right ventricle. As a result of the right to left shunt through the ventricular defect arterial oxygen saturation is below normal.

The anesthesiologist, in evaluating the patient with the pediatric cardiologist and the cardiac surgeon knows that there is no chance of long term survival without surgery if cyanosis is present. There perhaps is no ideal candidate but infants as young as one week of age are anesthetized for this palliative surgery even in heart failure. The perfect candidate would be compensated only slightly cyanotic devoid of an upper respiratory infection, at least four or five years of age and without other congenital defects.

The type of procedure to be done is not routinely a choice for the anesthesiologist to make. There is however, one exception—that being the very young desperately ill infant markedly cyanotic. This type of patient should not be subjected to a long difficult anastomosis such as a Blalock or Potts operation. Preference is a Brock operation that can be accomplished in a matter of minutes accepting the possible need for further surgery at a later date.

There are three generally accepted operations for palliation. The Blalock-Taussig is the surgical creation of a ductus. Ordinarily this is achieved by the anastomosis of the subclavian artery to the pulmonary artery. Potts modification is the formation of a ductus by a side to side anastomosis of the aorta and the pulmonary artery. Sellors and Brock recommended the removal of the obstruction either by resection of the infundibulum in the right ventricle or by incising the leaflets of the pulmonary valve.

It is agreed that any of the three procedures leaves much to be desired. Open cardiotomy with repair of the inter ventricular septal defect and obliteration of the hindrance to normal pulmonary circulation alone results in a cure.

The inherent difficulties in gaining free exchange of thoughts with an infant or child are complicated by the pampered or spoiled qualities imposed on the patient by anxious parents. The patient must be visited preoperatively so that at the time of surgery, at least one familiar face is visible.

Meperidine (Demerol) one milligram per pound of body weight and atropine sulfate, 0.1 mg (1/600 gr) to 0.2 mg (1/300 gr) are ordered ninety minutes prior to induction of anesthesia.

If the patient is not receiving oxygen therapy prior to receiving the hypodermic injection, he should be placed under a tent or croupette. Two reasons make this essential: the stimulus of the needle prick makes him cry (further decreasing the oxygen reserve) and the possible potential respiratory depression from the narcotic. The patient is then transported to the operating room receiving oxygen. If the child is apprehensive or poorly sedated, we use intramuscular pentothal sodium in a 5 per cent solution to supplement the preliminary medication.

A vein is cannulated after we attain first plane, third stage anesthesia. This is done after the initial induction of anesthesia in order to avoid pain and stress to the patient.

Infants, less than six months of age, are intubated prior to the administration of anesthesia. We believe this to be a more rapid means of securing adequate oxygenation and less harmful in the end. The technique of direct vision laryngoscopy and endotracheal catheterization while the patient is awake is not recommended for the novice anesthesiologist.

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Oxygen saturation of less than 80 per cent dictates supplementation by hypothermia and either oxygen alone being inspired and hypnosis provided by diluted, repeated intravenous doses of pentothal sodium or the use of cyclopropane and oxygen mixtures to provide the general anesthesia. The depth of anesthesia need never be greater than the first plane third stage, if respirations are controlled.

A clear-cut danger period exists from the induction of anesthesia to the termination of its course. We believe the anesthesiologist should stand so that it is possible to observe the heart action and the degree of inflation of the lung whenever the chest is open. Times of added stress are during the opening of the pericardium. Too great pressure on the reservoir bag will force the heart out at an acute angle. This results not from reflex, but from mechanical torsion of the heart on the great vessels which causes poor ventricular output, and hence asystole.

Ventriculotomy is attended by hemorrhage of varying degrees and the introduction of a probe into either the pulmonary artery or aorta precipitates hypotension instantaneously. The surgeon must wait until the anesthesiologist is satisfied with the pulse rate, systemic blood pressure, and ventricular output before attempting further intervention.

Still another trying time is the period spent closing the pericardial sac. Because more blood reaches the lungs following the Brock operation particularly if an immediate left to right shunt exists the left ventricle dilates. Extreme cooperation between surgeon and anesthesiologist must be exercised or the heart action will fail because of the effect of the tamponade. The blood pressure, pulse rate and left ventricular output must be closely observed for signs of inadequacy. Direct vision of the heart by the surgeon is inadequate to ascertain the early effects of the pericardial closure. He must rely upon his colleague the

Cyclopropane and oxygen have proven to be the most controllable agents for us. We prefer to use a non rebreathing technique, either by means of the Leigh Belton valve or the Fink non breathing valve. Respirations are assisted early in anesthesia and then controlled once a pneumothorax is present. Hypothermia is indicated in cyanotic heart lesions, but the temperature need not be reduced to less than 90° Fahrenheit (33° Centigrade). The important safeguard is the avoidance of heat retention and hyperpyrexia during the course of anesthesia and in the immediate (twenty four hours) postanesthetic period.

Children, six years of age or older, are anesthetized by slowly injecting thiopentothal sodium (Pentothal) 100 to 200 mg into a vein while oxygen by gravity and pressure is insufflated around the face. A muscle relaxant either decamethonium bromide 0.5 mg to 1.5 mg or succinylcholine hydrochloride 5 to 15 mg is injected intravenously. The larynx is visualized and one of three or four previously selected endotracheal catheters is introduced into the trachea. (We place on the anesthesia machine at least three noncuffed endotracheal tubes—one thought preanesthetically to be of the proper diameter then one the next size smaller and the third the next size larger than the estimated proper dimension.) Trauma to the larynx should be avoided and thus the danger of laryngeal edema can be minimized. Immediately following the insertion of the endotracheal tube by means of adaptors oxygen is supplied from a reservoir. The respirations are supported and then controlled by compression of the oxygen reservoir bag. Balanced anesthesia utilizing mixtures of four to five liters of oxygen and equal amounts of nitrous oxide have proven satisfactory in the older age group if the arterial oxygen saturation is above 80 per cent preoperatively. The preoperative oxygen tension is determined at the time of cardiac catheterization.

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anesthesiologist for his critical evaluation. Ordinarily the heart rate will be slow before it stops and by constant observation this is noted. Small doses of intravenous atropine sulfate, adequate oxygen intake and cessation of the manipulation of the heart will rapidly restore adequate function.

Blood should be replaced as lost, but care must be exercised not to exceed the volume lost. Polycythemia and increased blood volumes are present in most cases prior to anesthesia.

We suggest that a catheter be inserted through a nares into the stomach and allowed to remain in place throughout the course of anesthesia. Infants and children normally have no inhibition of gastric secretions during anesthesia. This affords the hazard of aspiration of secretions around the loosely fitting catheter in the larynx. Also the liquid is apt to loosen the adhesive tape securing the endotracheal catheter in place.

At times the mouth is so moist that we suture the endotracheal catheter in place rather than have it become dislodged. We realize the unesthetic appearance of this but the risk of inadvertent extubation or bronchial intubation far outweighs the disadvantages of a well placed suture in the corner of the mouth.

For a complete review of the technique of hypothermia the interested reader is referred to *Hypothermic Anesthesia* by Virtue.

Patients with a Tetralogy of Fallot are not ideal risks but the pleasure of offering a full life to an incapacitated infant is most gratifying for all participants.

PULMONARY STENOSIS

PULMONARY STENOSIS is a congenital abnormality in which there is an obstruction to the blood flow to the pulmonary artery. The malformation may be in the valve, or there may be infundibular stenosis in the right ventricle.

Although Taussig once wrote that pure pulmonary stenosis was rare, cardiac clinics throughout the world have observed pulmonary stenosis to be one of the common congenital defects of the heart. As a single lesion, pure valvular stenosis is more common than is the infundibular obstruction of the right ventricle. In patients with Tetralogy of Fallot the reverse is true—namely, the obstruction to the pulmonary flow is nearly always caused by infundibular obstruction.

Cyanosis may or may not be present, it depends upon the presence of a patent foramen ovale allowing a right to left shunt. The patient usually suffers more from fatigue than any other complaint. Growth development is usually normal and mental retardation practically never occurs.

Physical findings associated with pure pulmonary stenosis are cardiac enlargement (which is progressive), absent pulmonary second sound, harsh systolic murmur in the pulmonic area, and a systolic thrill over the obstructed area.

On x-ray examination the pulmonary vascular markings are normal. For the majority of patients the main pulmonary artery is dilated. The heart may be of normal size or enlarged. The right ventricle is prominent.

PULMONARY STENOSIS
CATHETERIZATION DATA

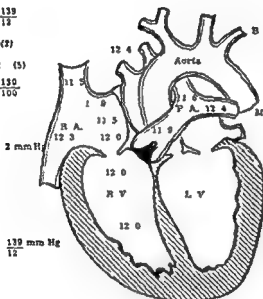
P A. $\frac{21}{8}$

R V $\frac{139}{12}$

R A. (2)

P V C (5)

B A $\frac{130}{100}$



B A. $\frac{130}{70}$

B A

Cont 11.2

Cap 16.8

Sat 86%

MPA $\frac{21}{8}$

Normal Values

Right Atrium 0-4

Right V ntricl $\frac{15-30}{0-2}$

Pulmonary Artery
 $\frac{15-30 (16)}{5-12}$

139 mm Hg
12

Oxygen Tensions Expressed in Volume per cent

All Pressures Expressed in mm of Mercury

O₂ Consumption 130 c/min.

Systemic Blood Flow 3.1 l/min.

Pulmonary Blood Flow 3.1 l/min

Shunts none

The electrocardiogram almost invariably shows right ventricular hypertrophy

Cardiac catheterization demonstrates a gradient in systolic pressure from pulmonary artery to right ventricle. The pressure in the chamber may be much higher than that in the systemic circuit. There is no evidence of a left to right shunt at any level. If there is a patent foramen ovale allowing a right to left shunt, arterial oxygenation is below normal.

Angiocardiography is not necessary for diagnosis and may be dangerous if the stenosis is severe.

Surgery is indicated when the stenosis is of significant severity, i.e., a gradient of 50 mm from pulmonary artery to right ventricle. The operation of choice is an adaptation of the Sellors Brock procedure, using a guillotine knife devised by Nichols. The pulmonary valve is opened by valvulotomy and the obstruction alleviated. The dangerous periods during anesthesia are the opening of the right ventricle with resultant hemorrhage, the initial probing into the pulmonary artery and right ventricle (at which time the ventricular output will be reduced), the danger of hemorrhage from a laceration of the pulmonary artery at the time of the achievement of the valvulotomy, and finally at the closure of the pericardium when the anesthesiologist must advise the surgeon of the effects of the closure.

Preliminary medication is utilized to retard the basal metabolic rate to inhibit untoward reflexes during intubation and to reduce the amount of general anesthetic agents needed. Meperidine is preferred as the narcotic for children and atropine sulfate for the abolition of reflexes.

Care must be exercised during intubation to avoid anoxia (even momentary) so that the heart will not be further belabored. The same meticulous attention to de-

PULMONARY STENOSIS
CATHETERIZATION DATA

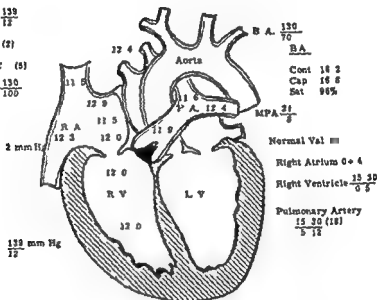
P A. $\frac{31}{8}$

R V $\frac{139}{12}$

R A. (2)

P V C (5)

B A. $\frac{130}{100}$



Oxygen Tensions Expressed in Volumes per cent

All Pressures Expressed in mm of Mercury

O₂ Consumption 130 c/min
Systemic Blood Flow 3.1 l/min.
Pulmonary Blood Flow 3.1 l/min
Shunts none

PATENT DUCTUS ARTERIOSUS

A PATENT DUCTUS ARTERIOSUS is the persistence after birth of the normal open fetal passage between the pulmonary artery and the aorta. Christie reported 65 per cent of ducti were patent at two weeks, 12 per cent at eight weeks, and 1 2 per cent after one year of life.

Patent ductus arteriosus has been likened to a time bomb by Burwell. Shapiro and Keys stated that 80 per cent of patients with this defect will succumb to it and that those who are alive at seventeen years will die at an average age of thirty five.

The symptoms are minimal when the ductus is small (4 mm or less in diameter) but increase in direct proportion to the diameter of the defect. Dyspnea and fatigue are usually the earliest symptoms, being manifestations of myocardial strain and pulmonary congestion.

Physical signs are usually quite classical. The persistence of a murmur from infancy heard in the pulmonary area, both systolic and diastolic is typical. The murmur has been described as 'machinery like' due to the persistent hum and as a 'to and fro' murmur because of its persistence in the cardiac cycle. A thrill can usually be palpated near the sternum in the second or third left inter space. The thrill can most easily be detected during ventricular systole. The systemic blood pressure is usually normal. However a tendency does exist for the diastolic pressure to be lower than normal, because of the existence of a fistula between the aorta and a low pressure chamber.

The x rays show the pulmonary vascular markings to be

tail is required to select endotracheal catheters of proper size preanesthetically as is needed during any pediatric anesthesia. Mixtures of cyclopropane and oxygen administered by means of to and fro carbon dioxide absorption technique or non rebreathing valves have proven most satisfactory. Ether is seldom used in our clinic for pediatric anesthesia as we prefer to avoid the increased amount of tracheobronchial secretions attendant on its use in light anesthesia. The essential requirements for anesthesia in patients with pulmonary stenosis are (1) adequate oxygenation (2) constant surveillance of the anesthetized patient (3) a light to moderate depth of anesthesia (first or second plane third stage) and (4) the use of actively assisted or controlled respirations from the time of intubation of the trachea until the chest wall closure is air tight.

PATENT DUCTUS ARTERIOSUS CATHETERIZATION DATA

Pressures Obtained

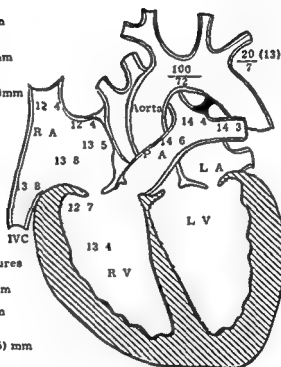
RA 0 mm

RV $\frac{20}{5}$ (10) mm

PA $\frac{20}{7}$ (13) mm

BA $\frac{120}{54}$ (80) mm

Aorta $\frac{100}{70}$ (92) mm



Aorta

Cont	17 8
Cap	17 8
Sat	98 9

BA

Cont	17 1
Cap	18 1
Sat	95 7

Normal Pressures

RA 0 + 4 mm

RV $\frac{15}{0+5}$ 30 mm

PA $\frac{15}{5}$ 30 (15) mm

Aorta $\frac{120}{70}$ mm

Oxygen Values expressed in Volumes per cent

Pressures expressed in mm of mercury

O₂ Consumption 138 6 cc/min

Systemic Blood Flow 3 5 l/min

Pulmonic Blood Flow 5 4 l/min

Shunts L to R 1 9 l/min

R to L 0

increased The main pulmonary artery is prominent The heart is as a rule of normal size although it may be enlarged If enlarged the left ventricle is prominent

The electrocardiogram is usually normal Left ventricular hypertrophy pattern appears if the shunt is great

On cardiac catheterization a left to right shunt is demonstrable at the level of the pulmonary artery Frequently the tip of the catheter will pass through the ductus and appear in the descending aorta Pulmonary artery pressure is normal except in those cases in which pulmonary vascular changes have increased resistance to flow These changes may be so marked that pulmonary pressure equals systemic and a reversal of flow through the ductus becomes possible

On thoracic aortography the pulmonary artery may become opacified when the contrast substance is swept around the region of the distal transverse arch

All cardiac anesthesiologists enjoy dealing with patent ductus arteriosus cases The mortality rate is roughly 1 to 2 per cent in the large cardiovascular centers Potts and McQuiston did more than 300 consecutive cases without a hospital mortality The mortality rate increases with the age of the patient after fourteen years of life Normally of all heart cases these are the most ideal candidates for anesthesia Only in the extremely young two or three week old infant is there need to anesthetize a patient with patent ductus arteriosus when heart failure or other complicating factors are present.

There is almost universal agreement that a right to left shunt through the ductus contraindicates surgery Other contraindications are bacterial endocarditis and upper respiratory infection

The only added strain placed on the cardiovascular team is the complete cheerfulness of the patient's family in subjecting a loved one to surgical therapy They know the

PATENT DUCTUS ARTERIOSUS CATHETERIZATION DATA

Pressures Obtained

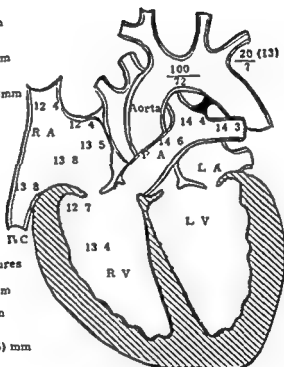
RA (0) mm

RV $\frac{20}{5}$ (10) mm

PA $\frac{20}{7}$ (13) mm

BA $\frac{120}{64}$ (80) mm

Aorta $\frac{100}{70}$ (92) mm



Aorta

Cont 17.6
Cap 17.8
Sat 98.9

BA

Cont 17.1
Cap 18.1
Sat 95%

Normal Pressures

RA 0 + 4 mm

RV $\frac{15}{0+5}$ 30 mm

PA $\frac{15}{8-12}$ 30 (15) mm

Aorta $\frac{120}{70}$ mm

Oxygen Values expressed in Volumes per cent

Pressures expressed in mm of mercury

O₂ Consumption 138 ± cc/min
Systemic Blood Flow 3.5 l/min
Pulmonic Blood Flow 5.4 l/min
Shunts L to R 1.9 l/min
R to L 0

mortality rate is low, but if a fatality occurs in their own immediate family, to them the rate is 100 per cent

There is no unusual requirement preoperatively other than the routine preanesthetic visit by the anesthesiologist. Preliminary medication is directed toward the criteria advocated by Adrian: (1) psychic sedation (2) the reduction of the metabolic rate for which we prefer meperidine (Demerol) 1 mg per pound of body weight—not to exceed 100 mgs (3) to abolish secretions from the respiratory tract and (4) to avoid anticipated undesirable effects by vagal stimulation atropine sulfate has been our drug of choice to accomplish these two last inhibitory effects

Children (younger than fourteen years of age) are anesthetized in the operating room and intubated prior to doing a venous cannulation. Infants from one month to six years of age are anesthetized with a cyclopropane oxygen mixture. Children older than six years, are handled differently. Hypnosis is induced by the intravenous administration of pentothal sodium. Facilitation of laryngoscopy and endotracheal intubation is produced by the intravenous administration of short acting curare like drugs such as decamethonium bromide or succinylcholine hydrochloride. Analgesia is produced by equal mixtures of oxygen and nitrous oxide.

All patients should be intubated. This will afford an artificial airway that will allow the chest cavity to be entered without untoward physiological response from the existence of a pneumothorax. Respirations should be either controlled or effectively assisted by manual compression on the breathing bag.

Watterman, Samson and Bailey acting as a committee for the Section on Cardiovascular Surgery of the American College of Chest Physicians reported on 4448 cases of patent ductus arteriosus that were operated on in the

United States and abroad Five hundred fifty two cases were eliminated because of insufficient data, leaving 3,896 that fulfilled the rigid criteria imposed by the committee Two thousand nine hundred twenty nine operations were performed on children and 967 on adults The results were a mortality rate of 2.3 per cent in the patients younger than fourteen years of age, and 5.5 per cent in the older group (above fourteen years of age) Of the survivors, the clinical results in the children were 98.3 per cent satisfactory and 95.5 per cent satisfactory for the adults Forty four deaths were included that occurred in the hospital Of this number (44), twenty nine were due to hemorrhage at operation six were listed as cardiac asystole, two from pulmonary edema two as a result of cerebral hemorrhage two from hyperpyrexia (probably hypoxia) and one each from ventricular fibrillation embolization (not localized) and unknown cause

A brief review of this report shows the greatest single danger during the anesthesia and surgery to be hemorrhage This points out very clearly that although the overall mortality rate is low, extreme care must be taken to have a patent large bore cannula inserted into a vein so that blood loss can be quickly replaced if hemorrhage should occur during surgery

A most important clinical evaluation must be made by the anesthesiologist when the ductus is occluded Normally the diastolic pressure is elevated immediately following the occlusion, as the fistula is interrupted If there are untoward effects produced by the occlusion, they will be manifest within five to ten minutes Myocardial insufficiency is the chief factor in the production of symptomatology preoperatively Hypotension pulmonary edema cyanosis increased venous pressure or tachycardia occurring at the time of or immediately after, occlusion of the

patent ductus arteriosus indicate the need to abandon the operation. The anesthesiologist must be aware of these responses and inform the surgeon if any of the ill effects are observed.

The salient points for the anesthesiologist to recall are (1) proper preliminary medication (2) the need to intubate, (3) the adequate control of respiration (4) utilization of a high concentration of oxygen (5) proper evaluation of the physiological responses at the time the ductus is clamped and (6) the inherent danger of hemorrhage.

COARCTATION OF THE AORTA

COARCTATION of the aorta is a congenital defect in which there is a stricture in the aorta. Most frequently the narrowing of the aorta occurs just below the origin of the left subclavian artery. The decrease in size of the lumen prevents an adequate supply of blood from reaching the lower half of the body. The stricture may vary in size but sooner or later, strain will be imposed on the heart.

There are seldom early symptoms of coarctation of the aorta. The complaints of headache, dizziness, dyspnea and blurred vision are a result of the associated hypertension. The decreased flow to the lower extremities is responsible for the numbness and coldness of the legs and feet.

The average age of death in untreated cases has been reported by Abbott to be thirty-two. There can be no question about the lesion being malignant if untreated.

A definite sign of coarctation of the aorta is hypertension in the upper extremities with hypotension in the lower extremities. If bilateral femoral artery pulsations are absent, coarctation of the aorta should always be suspected.

X-ray examinations frequently reveal evidence of the collateral circulation around the defect. The intercostal arteries become vastly overloaded and erode into the rib edges producing the so-called notching of the ribs.

Aortography is an aid in confirming the diagnosis and gives an index as to the length of the constriction in the aorta.

The optimum age for surgical intervention is between six and twelve years of age. The mortality and morbidity rates are increased in the older patients due to early atherosclerosis of the aorta and chronic renal failure. Children, less than six years of age, are seldom operated upon because of the inherent risk of the diameter of the anastomosed aorta not enlarging as the child's size increases. Adults over thirty years of age are poor candidates because of the calcific changes in the wall of the aorta which predisposes to the formation of aneurysms and poor healing quality.

A general guide as to the ideal age for surgical intervention may be outlined as (1) age between six and twelve years with the upper limit of thirty years (2) evidence of hypertension in the upper extremities (3) cardiac enlargement of slight to moderate degree and (4) absence of subacute bacterial infection.

One recent development that has increased the beneficial surgical results has been the use of grafts. Gross has demonstrated the long term efficiency of preserved homografts in the surgical correction of coarctation of the aorta.

Preliminary medication is directed toward reducing physical and psychic strain and the lessening of cardiac irritability. Meperidine (Demerol) 1 mg per pound of body weight is ordered sixty to ninety minutes prior to the scheduled time of anesthesia. We never exceed a dose of 100 mg regardless of the weight or age of the patient. Atropine sulfate is preferred as the vagal inhibitor, and the dosage is varied according to generally accepted standards of age and body weight.

Patients older than ten years of age have two veins cannulated after receiving the preliminary medication but prior to the induction of anesthesia. Two avenues of parenteral fluid administration are chosen because of the blood loss attendant upon the execution of the thoracic

cotomy The tremendous proliferation of the collateral circulation and the increase in the size of all blood vessels in the chest makes hemostasis a difficult and time-consuming ordeal Whole citrated blood should be administered intravenously just prior to the induction of anesthesia It is easier to stay ahead of blood loss rather than place the added strain of hemorrhagic shock on a belabored cardiovascular system

The choice of anesthetic agents is usually limited to non inflammable drugs so that electrocoagulation may be used by the surgeons to further minimize blood loss We prefer to use 50 to 300 mg of pentothal sodium intravenously, followed in the same route by 20 to 50 mg of succinylcholine hydrochloride The largest cuffed endotracheal catheter that can be inserted without trauma is introduced under direct vision The lungs are inflated with oxygen by manual compression of the rebreathing bag for at least five minutes The patient is then turned onto the right side in the lateral decubitus position Respirations are controlled, the systemic blood pressure is ascertained in the lateral position, and if normal (or not less than 20 mm of mercury less than in the supine position) nitrous oxide is added to the oxygen in the reservoir bag We prefer mixtures of nitrous oxide three liters and oxygen two liters The concentration of inspired oxygen should never be less than 35 volumes per cent in any anesthesia for cardiac surgery

Intravenous pentothal sodium is added as needed to produce hypnosis Analgesia is afforded by the nitrous oxide and oxygen mixture Succinylcholine hydrochloride may be used in 0.1 per cent or 0.2 per cent solution if needed to facilitate the control of respirations and aspiration of the tracheobronchial tree

The systemic blood pressure will be elevated at the time the aorta is clamped proximal to the coarctation The

hypertension will remain exaggerated until after the anastomosis has been completed and the clamps removed. Immediately after the channel has been opened the systemic blood pressure will be lowered sometimes as much as 100 mm of mercury. If it remains low for more than five minutes, a dilute solution of a vasopressor should be used. There are several physical factors that control the systemic blood pressure. They are (1) elasticity of the arterial walls (2) circulating blood volume, (3) pumping action of the heart (4) viscosity of the blood and (5) peripheral resistance. The hypotension immediately evidenced after the surgical correction of the coarcted area of the aorta is due to the tremendous change in the lack of peripheral resistance. Blood simply surges into the lower half of the body for the first time in the patient's life.

As time elapses the hypertension will return for varying periods of time. ordinarily the blood pressure is at a normal level two weeks after surgery.

It is the author's personal belief that hypotensive drugs have no therapeutic place in the course of anesthesia for patients with heart damage and we further believe it to be a dangerous way to manage a patient with coarctation of the aorta.

Blood should be replaced as need be to maintain a normal circulating blood volume. Blood loss is manifest during the incision through skin and muscles of the chest. Hemorrhage can occur from intercostal arteries or a laceration of the wall of the aorta.

The mediastinum can best be made free of motion that impedes the surgery by use of controlled respirations.

In general the major problems of an anesthesiologist during the course of anesthesia for coarctation are blood loss and the establishment and maintenance of a quiet chest to facilitate surgery.

ATRIAL SEPTAL DEFECTS

ATRIAL SEPTAL DEFECTS are among the most common of isolated congenital malformations of the heart. Probably they are second only in frequency to ventricular septal defects.

There are two general classifications of interatrial septal defects: ostium primum and ostium secundum. Our series of one hundred operated cases revealed ostium primum to have been present in seventeen cases and ostium secundum in eighty-three instances. There was a 3:1 ratio of women to men. Fifteen patients had a partially anomalous pulmonary venous drainage; eight had a Lutembacher syndrome (atrial septal defect and mitral stenosis) that was corrected in a single surgical procedure.

An atrial septal defect is compatible with a relatively normal life in some; others may die in infancy or childhood from congestive failure. The average life expectancy is thirty to thirty-five years.

The symptoms vary with the degree of increased pulmonary blood flow. Typically patients complain of fatigability, progressive exertional dyspnea, paroxysmal tachycardia, chest pain and congestive heart failure.

Physical findings are a systolic murmur heard in the second or third interspace to the left of the sternum, an accentuated pulmonic second sound and a diastolic murmur in the pulmonic area or over the apex. The systemic blood pressure is usually within normal variations. The auscultatory systemic blood pressure is usually 10 to 15 mm of mercury higher than the palpatory determination.

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ATRIAL SEPTAL DEFECT

CATHETERIZATION

DATA

Pressures Obtained

PVC (0)

BA $\frac{128}{76}$

PA $\frac{38 (23)}{14}$

EV $\frac{38}{3}$

RA (0)

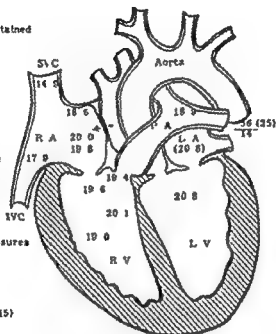
(1)

Normal Pressures

RA (0)

RV $\frac{15-30}{0-5}$

PA $\frac{15-30 (15)}{5-12}$



BA

Cont 20.8

Cap 22.7

Sat 92%

All Pressures Expressed in mm of Mercury

Oxygen Values Expressed in Volumes per cent

Pulmonic Blood Flow 20.7 l/min

Systemic Blood Flow 6.1 l/min

Shunts L to R 18.8 l/min

R to L 0

Overall 14.6 l/min

O₂ Consumption 269.1 l/min

The diagnosis of atrial septal defect is usually made following right heart catheterization. There are two main guides in the cardiac-catheterization laboratory that enable the diagnosis to be made: the passage of the catheter from the right atrium to the left atrium, and an oxygen tension of two volumes per cent or greater in the right atrium than the venous blood samples of inferior and superior vena cava.

Seventy-eight cases of atrial septal defects had preoperative right heart catheterization. The data revealed an increase in pulmonic blood flow in all cases with variations between six to thirty-one liters per minute. The average right to left shunt was sixteen hundredths (0.16) of a liter per minute. Pulmonary hypertension varied from thirty mm of mercury (30 mm) to one hundred mm of mercury (100 mm).

Roentgenograms show characteristically increased pulmonary vascular markings: a large main pulmonary artery, large right and left branches of the pulmonary artery, and right heart enlargement.

The electrocardiogram is usually abnormal. Normal sinus rhythm is present but with right ventricular hypertrophy and right bundle branch block.

The anesthesiologist in evaluating the patient with the other members of the team uses the following general guide for the ideal candidate as (1) age between fifteen to thirty years, (2) ostium secundum atrial defect, (3) moderate cardiac enlargement, (4) the shunt from left to right (without evidence of cyanosis), (5) pulmonary hypertension—not to exceed one half of the systemic blood pressure, and (6) free of cardiac decompensation. It is obvious that ideal requirements can not always be adhered to, but reverse shunts, age of fifty-five years or greater, and markedly elevated pulmonary hypertension are contraindications to successful anesthesia.

form agent and thiopentothal sodium intravenously. An endotracheal catheter with inflatable low pressure cuff of proper dimension is introduced under direct vision. The lungs are inflated with oxygen and the blood pressure and pulse ascertained following intubation. Ordinarily, the systemic blood pressure will be slightly elevated following intubation of the trachea.

Surgery is performed with the patient in the supine position by means of a right thoracotomy.

Equal mixtures of nitrous oxide and oxygen are used to allow adequate oxygenation and analgesia. Hypnosis is provided by repeated intravenous injections of small amounts of thiopentothal sodium. Respirations are controlled until after the chest wall approximation is airtight and a pneumothorax no longer exists.

Arrhythmias are prone to develop during the surgical closure of the septal defect due to suturing of the atrial walls. The irregularities are of atrial origin such as atrial premature systoles, atrial flutter and atrial fibrillation. We make no attempt to digitalize the patient during anesthesia if these irregularities occur, unless evidence of heart failure follows their appearance. There are only two ways it is possible to achieve rapid digitalization, intravenous quinidine hydrochloride and intravenous Deslanoside D (Cedilanid D). Intravenous quinidine therapy is very dangerous. Di Palma has reported deaths following this route of administration with doses as small as 100 mg. Quinidine should not be used intravenously unless constant electrocardiographic tracings are being made and interpreted. The digitalizing dose of Cedilanid is 1 mg.

There is a characteristic syndrome following superior vena caval obstruction and the superior vena cava may be obstructed during the closure of an atrial septal defect. The obstruction may be extrinsic from pressure of an as

There are three different anesthetic adjuncts in use at the present time (1) hypothermia with open cardiectomy (2) a heart lung apparatus to allow open cardiectomy, and (3) the closed technique utilizing normal body temperatures. We have used all three of these techniques and prefer the closed technique at normal temperature. The mortality rate is low (12 per cent) it requires less time for anesthesia and all but two in one hundred cases were closed satisfactorily. Virtue has published his work on *Hypothermic Anesthesia*, and the anesthesiologists that use hypothermia are referred to his book for complete details of technique.

The patient is visited preoperatively by the anesthesiologist and questioned as to any untoward effects from the preliminary medication used for the cardiac catheterization. Pentobarbital sodium (Nembutal) grains $1\frac{1}{2}$ (100 mg) is ordered to be taken orally at the hour of sleep. Meperidine hydrochloride (Demerol) 50 mg to 100 mg depending on the patient's weight and general stamina is ordered to be given with atropine sulfate 0.4 mg (grains $1/150$) by hypodermic injection one hour (sixty minutes) before the scheduled hour of anesthesia.

The patient must have two veins cannulated prior to the induction of anesthesia. Infiltration anesthesia following the preliminary sedation is adequate. The patient is brought to the operating room and the electrocardiograph leads are placed in the proper position. The patient should have continuous electrocardiographs made during the closure of the atrial septal defect so that any ligature around the conduction system can be noted prior to being secured.

The systemic blood pressure recordings, pulse rate and respiratory rate are ascertained and recorded on the anesthesia chart.

Anesthesia is induced by utilizing a rapid acting curari

form agent and thiopentothal sodium intravenously. An endotracheal catheter with inflatable low pressure cuff of proper dimension is introduced under direct vision. The lungs are inflated with oxygen and the blood pressure and pulse ascertained following intubation. Ordinarily, the systemic blood pressure will be slightly elevated following intubation of the trachea.

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There is a characteristic syndrome following superior vena caval obstruction and the superior vena cava may be obstructed during the closure of an atrial septal defect. The obstruction may be extrinsic from pressure of an as

sistant's hand, or gauze retractor or intrinsic from acute surgical occlusion. The syndrome is the same: there is first cyanosis of the head, face, and upper extremity followed by marked venous distension of the upper extremities. The intravenous infusion rate is slowed or stopped, depending on the degree of occlusion. Whatever the cause and effect of the mechanism, the stasis should be brought immediately to the attention of the surgeon. It is essential to bear in mind that drugs and blood administered intravenously have no effect until after the occlusion is corrected. Blood loss is usually minimal following the closed technique and usually uneventful.

The actual arrhythmias may continue after surgery for one or two months but seldom for longer periods of time. The preoperative symptoms are corrected by surgery as there is a reduction of the pulmonic blood flow and a more adequate cardiac output.

MITRAL STENOSIS

MITRAL STENOSIS is usually the result of rheumatic valvulitis although rare cases of congenital stenosis of the mitral valve may occur and cases have been reported

Commissurotomy for mitral stenosis is the most frequently performed operation on patients with acquired heart lesions and accounts for more than one half of all heart operations. Members of the Department of Anesthesiology of Hahnemann Hospital in Philadelphia, Pennsylvania, have anesthetized more than 2,000 patients for this surgical procedure.

The symptoms and physical findings in patients with mitral stenosis are a result of the blockage or damming effect of the narrowed mitral valve orifice. This results in a backward type of congestion in the left atrium, pulmonary capillary bed, pulmonary artery, and right ventricle.

We believe the symptomatology to be so constant that on ward rounds, with no further information than the personal questioning, the diagnosis can more frequently than not be made. The most characteristic symptoms are in the order of occurrence: **SHORTNESS OF BREATH** after slight physical exertion or emotional stress—(this is due to an increase in pulmonary capillary pressure and a decrease in left ventricular output); **DYSPNEA** which occurs as the pathological changes progress and the cardiac output is further decreased (a result of elevated pressure in the left atrium, pulmonary capillary bed, pulmonary artery, and right ventricle); **HEMOPTYSIS** (blood spitting or flecks

of blood in mucus that is expectorated) is coexistent with further progression in the abnormal hemodynamics following a smaller diameter of the obstructed mitral valve with more impedance of the blood from the left atrium to the left ventricle

The most alarming of the cardiorespiratory symptoms is pulmonary edema. A history of a complaint relative to arterial embolization is present in about one quarter of all cases surveyed prior to anesthesia. The occlusion may have been to an extremity, to the kidney, lung or to the brain. This is very significant since insufficiency of a valve does not predispose to arterial embolization.

The predominant physical findings are a sharp mitral first sound, a mid to late diastolic murmur heard at the apex of the heart, a palpable thrill over the mitral valve, increased venous pressure, hypotension and atrial fibrillation. Blood pressure determinations deserve special attention since there is a most consistent variation of the accepted teaching, namely the palpatory systolic recording is higher by 10 to 15 mm of mercury than is the highest systolic level heard during auscultatory determinations of the systemic blood pressure in all individuals handicapped by mitral stenosis.

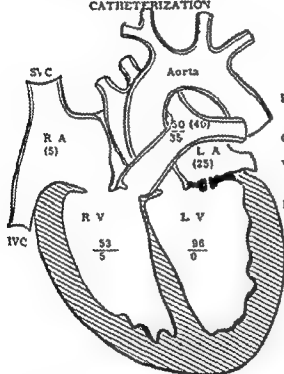
Hypotension as a preanesthetic complication exists if the palpatory systolic pressure is 100 mm of mercury or less. We have observed hypotension in 75 per cent of all patients during the examination period before surgery. The blood pressure findings are noted by both palpation and auscultation on the anesthesia records in the supine position and in the lateral position (patient turned onto the right side). Particular attention should be drawn to the rare patient with hypertension and mitral stenosis. Hypertension is defined as a systemic systolic blood pressure of 150 mm of mercury or greater or a systemic diastolic

MITRAL STENOSIS

SIMULTANEOUS PRESSURE DATA

RIGHT AND LEFT HEART

CATHETERIZATION



Pulmonary Capillary Pressure (22)

Cardiac Index 2.3 l/min

Ventricular Filling Gradient (20)

Mitral Valve Flow 125 cc

Mitral Valve Area $\approx 3 \text{ cm}^2$ All Pressures Expressed in mm of Mercury
Normal PressuresRight Atrium ≈ 4

Left Atrium (10)

Right Ventricle $\frac{15}{0} \frac{30}{+5}$ Left Ventricle $\frac{120}{0} \frac{10}{10}$ Pulmonary Artery $\frac{15}{5} \frac{30}{12}$ (15)Aorta $\frac{120}{70}$

Pulmonary Capillary Pressure 5-12

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in congestive failure, and (7) have cardiac enlargement of moderate degree only

Atrial fibrillation, arterial embolization, recent congestive failure, multivalvular involvement and cardiac enlargement are not contraindications to anesthesia and surgery however, the results are more apt to be palliative, than curative

Absolute contraindications are septicemia gallop rhythm intractable failure marked pulmonary fibrosis and marked cardiomegaly

The anesthesiologist should visit the patient preoperatively several times This will afford him the opportunity to judge whether or not the patient's medical progress is satisfactory Also a more clear-cut indication of survival can be obtained A guide to the patient's psychological attitude is more easily ascertained in three visits than by one preanesthetic evaluation

Dyspnea is a prime symptom of all patients with mitral stenosis and right time selection is essential to provide a rested patient for the ordeal of anesthesia and surgery We prefer pentobarbital sodium (Nembutal) grains $1\frac{1}{2}$ (100 mg) orally at the hour of sleep We use this for two or three consecutive nights prior to anesthesia, this allows in individualization of patient's responses

Secobarbital is ordered for oral ingestion one hour prior to induction in accordance with the effective palpatory systolic blood pressure as outlined in the chapter on preliminary medication If the palpatory systolic pressure is less than 90 mm of mercury no barbiturate is ordered for the day of anesthesia

Atropine sulfate 0.65 mg (1/100 gr) is ordered if the pulse rate is 60 per minute or less 0.4 mg (1/150 gr) if the pulse rate is between 60 and 80 per minute if more rapid than 80 per minute 0.3 mg (1/200 gr) is ordered one hour prior to anesthesia

blood pressure of at least 100 mm of mercury. We have observed hypertension in only fifty nine patients to date. It is noteworthy to report that there were nine deaths in the fifty nine cases, a mortality rate of 15.3 per cent. Our own over all hospital mortality rate has been 7.2 per cent. Thus those with hypertension have a doubled mortality rate. The unnatural lateral position further decreases the cardiac output. However, if the surgery is to be achieved with the patient in this position, the base line, or normal finding is determined at least twenty four hours prior to anesthesia and is used as a guide in selecting preliminary medication.

Right heart catheterization reveals an elevation in the pulmonary artery pressure, with a compatible elevation of the right ventricular pressure and a compensatory elevation of the pulmonary venous capillary pressure. There is a decrease in the cardiac output.

Left heart catheterization will show a pressure gradient from the left atrium to the left ventricle if mitral stenosis is present.

Roentgenographic examination will demonstrate an enlargement of the left atrium, a fullness of the pulmonary conus, straightening of the left heart border, and enlargement of the pulmonary arteries. Frequently the calcified mitral valve is visualized. Electrocardiographic tracings reveal atrial fibrillation in 50 per cent of the cases, right ventricular hypertrophy, and digitalis effects are nearly always constant.

The combined conference with the cardiologist, surgeon, and anesthesiologist should be the time of patient selection. The ideal candidate for mitral commissurotomy should be (1) less than fifty years of age, (2) free of acute rheumatic activity, (3) have signs of progressive mitral stenosis, (4) have a palpatory systemic blood pressure of 100 mm of mercury, (5) have a pure mitral stenosis, (6) not be

to accomplish atriotomy of the left atrium, we now prefer the supine position. The systolic blood pressure is more easily maintained between 80 and 100 mm of mercury or higher. This is further undisputable proof of the decreased left ventricular output produced by the lateral position and the additional handicap of exaggerated hypotension from an abnormal position.

A systemic systolic pressure of 80 mm of mercury must be considered to be the critical lower acceptable blood pressure.

It occasionally happens that hypotension results in the supine position. In such an instance surgery is cancelled, the patient is awakened as rapidly as possible to full consciousness, and sent to a recovery room. Actually judicious interruption of plans is advantageous in some instances; it makes the families all the more familiar with the inherent risks and proves conclusively the extreme interest of the team to provide the best known care for the patient. This has always been welcomed by the relatives and candidates. To the best of the author's knowledge no candidate has refused to be subjected to the induction of anesthesia on a subsequent date. The importance of prompt cancellation when hypotension exists with the resultant depressed physiological status cannot be over emphasized.

It is preferable to control hypotension by physiological means such as allowing cardiac action to be uninhibited by traction or pressure. Sometimes this can not be achieved and the low pressure must be combatted pharmacologically. Various means are available and a common fault is to use whole citrated blood incorrectly. It must be borne in mind that the blood volume is slightly increased normally in patients with rheumatic heart disease. Blood volume is increased above normal limits if any evidence of congestive failure is present and finally the morbidity and mortality of patients with elevated total blood volumes

Two veins should be cannulated under infiltration anesthesia prior to being brought to the operating room suite. One should be in an arm (the dependent one if the lateral position is to be used) and the other cannula in a lower extremity.

Decamethonium bromide 2 to 4 mg is injected intravenously followed by 200 mg to 350 mg of thiopental sodium by the same route. The patient is intubated under direct vision laryngoscopy and the lungs inflated with pure oxygen. If following intubation and oxygenation of the patient the systemic systolic blood pressure is 100 mm of mercury or greater nitrous oxide is added so that a 50:50 mixture of oxygen and nitrous oxide is obtained. Whenever the pressure of the systemic circulation is less than 100 mm of mercury oxygen alone is used for inspiration. The patient is kept quiet by manual control of respiration and small repeated intravenous injections of thiopental sodium. Unquestionably a respiratory alkalosis will result but we think this to be helpful rather than harmful in requiring smaller amounts of anesthetic agents or hypnotics. Doctor Ivan Magill of London has said that the depth of anesthesia is too great when the anesthetist winks at the patient and the patient does not wink back.

Brachial plexus palsies may follow improper positioning of the patient's arms. We prefer to use an arm board for the arm containing the cannula and to have the other arm positioned so that it is flexed at the elbow with the wrist at the head but without producing tension on the brachial plexus. If neuropathy does occur judicious negligence will allow complete recovery to occur within four to six weeks.

The first 1000 cases anesthetized for commissurotomy at Hahnemann Hospital in Philadelphia were operated on in the lateral decubitus position. Since it is now known to be possible to gain access to the mitral valve with a right thoracotomy and dissection of the inter atrial grooves and

faith in his team will wait, with varying degrees of patience, until a more physiological period has been attained before attempting further intracardiac manipulation

The brain must be protected during intracardiac surgery against embolism. Embolization has been a complication during anesthesia only from the stenotic valves. Since mitral stenosis is the lesion most frequently encountered during anesthesia for heart surgery, this complication will be observed most frequently after mitral commissurotomy. The most satisfactory method of preventing this accident is to isolate the innominate artery and the left common carotid artery, both of which are occluded during the opening of the atrium and valvular manipulation if the left lateral thoracotomy is the entrance to the mitral valve. Time again must be called aloud so that the brain will not be deprived of oxygenated blood for more than one minute. Again the cerebral arterial blood supply should not be interrupted for at least twice the period of deprivation to the brain. No protection against cerebral embolization is used if the surgery is being accomplished with the patient in the supine position.

Respirations are controlled from the moment following intubation until the chest wall approximation is air tight. Respiratory paralysis, or apnea, has not been a problem to us. Voluntary respiratory activity will return within one to two minutes following the cessation of controlled respirations.

We endeavor to have the patient awake and responsive at the completion of surgery.

It would be ridiculous and presumptive to state that the technique outlined is the only acceptable way to conduct anesthesia for the surgery of mitral stenosis, but this has proven satisfactory in our hands since 1948. We are certain that time will bring further necessary advances.

are markedly increased. Thus blood parenterally is indicated solely as replacement to actual loss during surgical intervention. Of the numerous vasopressors available today, we prefer methoxamine hydrochloride (Vasoxyl), or Levarterenol bitartrate (Levophed). Methoxamine hydrochloride is used as the pressor agent if the pulse rate is greater than ninety per minute.

Each solution is prepared by adding 2 ml of the stock solution to a flask containing 500 ml of 5 per cent glucose and distilled water. In desperate extremes desperate measures of resuscitation are indicated. If no response is elicited by the two above named drugs, a solution of 3 ml of 1/1000 epinephrine in 500 ml of 10 per cent dextrose and distilled water is administered at a sufficient rate intravenously to maintain an adequate blood pressure.

One of the most vital functions of the anesthesiologist is to make the surgeon acutely aware of time. It is obvious that during the actual manipulation required to open a stenotic valve the ingress of oxygenated blood from the left atrium to the left ventricle is stopped so that there is no left ventricular output. Twenty seconds is the upper safe limit of complete occlusion. The seconds are counted aloud by the anesthetist in increments of five seconds after the valve has been obstructed for ten seconds. The surgeons have, on rare occasions, exceeded the safe time limit to sixty seconds and have been plagued by ventricular fibrillation as a result of anoxia in the coronary arteries and ventricles. Realizing full well that the heart has no reservoir for oxygen storage none the less we strongly request that the heart be rested for at least twice the period of time the valve was occluded.

The surgeon and the anesthesiologist should not attempt to keep secrets from one another. If hypotension is present, the surgeon should be informed and the exact blood pressure determination announced. A reasonable operator with

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blood pressure is ordinarily 10 to 15 mm of mercury higher than the auscultatory determination

Laboratory results are quite typical—there is a pressure gradient between the left ventricle and the aorta. Goldberg believes the pressure gradient is significant when it is 40 mm of mercury or more. The electrocardiographic tracings reveal a left axis deviation. Roentgenograms show two rather characteristic findings—one a concentric enlargement of the heart and the other a calcified aortic valve. Blood volume determinations are increased above normal if any evidence of decompensation exists.

The anesthesiologist in evaluating the patient has the following outline for an ideal candidate: (1) age less than fifty years; (2) pure aortic stenosis; (3) absence of acute rheumatic fever; (4) cardiac enlargement of only moderate degree; (5) no evidence of hepatomegaly, dependent edema, or pleural effusion; (6) Class I, II, or III functional classification of the American Heart Association; and (7) a palpatory systolic blood pressure greater than 100 mm of mercury.

There is a simple test that has proven very useful to the author in addition to the ideal findings outlined above—the simple procedure being to ask the patient to hold his breath as long as possible. If the pulse rate is markedly increased following the voluntary transitory apnea or angina of moderate to severe nature follows breath holding of at least thirty seconds the candidate has poor myocardial reserve and probably will not tolerate even a light plane of anesthesia.

Two operations are accepted to relieve aortic stenosis: (1) the early transventricular approach advocated by Bailey in 1952 and (2) and a retrograde admittance via the aorta distal to the stenotic aortic valve. Each operation has certain advantages and disadvantages to the anesthesiologist.

AORTIC STENOSIS

AORTIC STENOSIS may be congenital or a result of either arteriosclerosis or rheumatic valvulitis. The defect may exist alone as a pure aortic stenosis or with varying degrees of aortic insufficiency. It is also frequently encountered with mitral valve disease and as such is a complicating factor.

In aortic stenosis the tricuspid valve of the aorta fails to open properly during the left ventricular systole. This produces an obstruction to the outflow tract and results in left ventricular hypertrophy.

The symptoms are progressive since aortic stenosis is a completely intractable condition and is characteristically unresponsive to medical therapy. The lack of response to medical therapy is due to a combination of factors: there is a serious functional interference with the mechanics of coronary artery filling and progressive hypertrophy of the left ventricle. The cardinal symptoms are syncope produced by a narrow pulse pressure, angina which is a result of inadequate coronary artery filling, fatigue from poor ventricular output, dyspnea upon exertion which results from poor myocardial tone and dependent edema.

Physical signs are quite characteristic: (1) a harsh systolic murmur heard over the second interspace to the right of the sternum; (2) there is an absent second sound in the aortic area in 96 per cent of patients; (3) the systemic systolic blood pressure is low with a high diastolic blood pressure; (4) the pulse is usually regular with atrial fibrillation almost never encountered; (5) the palpatory systolic

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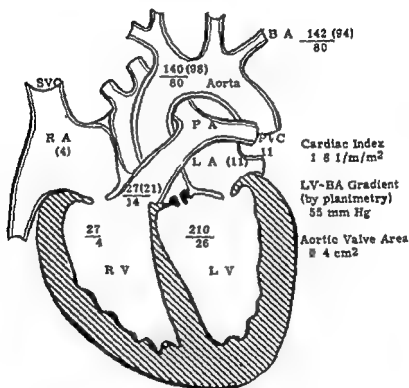
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AORTIC STENOSIS

SIMULTANEOUS PRESSURE DATA

RIGHT AND LEFT HEART

CATHETERIZATION



All Pressures Expressed in mm of Mercury
Normal Pressures

Right Atrium	$0 + 4$	Left Atrium	(10)
Right Ventricle	$\frac{15}{0 + 5}$	Left Ventricle	$\frac{120}{\approx 10}$
Pulmonary Artery	$\frac{15}{5-12}$	Aorta	$\frac{120}{70}$

Pulmonary Capillary Pressure 5-12

which should be mentioned Transventricular aortic commissurotomy has the distinct advantage of being rapidly achieved with a resultant decrease in total time of anesthesia The disadvantages are (1) high incidence of ventricular fibrillation precipitated by occlusion of the stoma coronary arteries or by a trigger mechanism instituted by probing against the interventricular septum, (2) hemorrhage from the opening made in the left ventricle and (3) the hypotension produced by positioning the patient in the lateral position The advantages of the supra aortic valve approach are (1) no hypotension from positioning of the patient (2) relative freedom from ventricular fibrillation as a result of instrumentation of the valve and (3) resulting in a more functional valve

The anesthesiologist should visit the patient several times preoperatively during the visits the blood pressure should be ascertained and any variation in the incidence or degree of angina noted Thus a more rational approach to preanesthetic medication can be obtained

Nearly all patients with aortic stenosis have difficulty sleeping at night We sedate them with pentobarbital sodium (Nembutal), grains $1\frac{1}{2}$ (100 mg) orally and meperidine 50 to 75 mg by hypodermic injection at the hour of sleep Secobarbital grains $\frac{3}{4}$ (50 mg) orally and atropine sulfate grains $\frac{1}{150}$ (0.4 mg) by hypodermic injection is ordered to be given one hour (sixty minutes) before the time of anesthesia

The patient must have two veins cannulated prior to the induction of anesthesia The wrist in which the radial pulsations are feeblest is chosen for the upper extremity One cannula should be introduced into an ankle vein

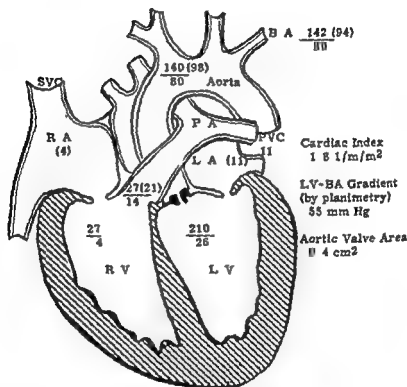
When the nurses tray is set up and the surgical team properly gloved and gowned, anesthesia is induced It is advisable to place an oxygen mask over the patient's face

AORTIC STENOSIS

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RIGHT AND LEFT HEART

CATHETERIZATION



All Pressures Expressed in mm of Mercury
Normal Pressures

Right Atrium	0 ± 4	Left Atrium	(10)
Right Ventricle	$\frac{15 \ 30}{0 \pm 5}$	Left Ventricle	$\frac{120}{0 \ 10}$
Pulmonary Artery	$\frac{15 \ 30 \ (15 \ 18)}{5 \ 12}$	Aorta	$\frac{120}{70}$

Pulmonary Capillary Pressure 5 12

The anesthesiologist must inform the surgeon when the cardiac action and the vital signs of the patient will allow the aortic valve to be manipulated. The valve can not be occluded for more than ten to twenty seconds at one time. The surgeon should wait until an adequate circulation exists for several minutes (two to three minutes) before initiating further surgical intervention. He must heed the anesthesiologist's advice and the anesthesiologist should have sufficient command of the anesthetized patient's status so that he can give the desired information instantly.

The arrhythmias most frequently encountered are premature ventricular systoles, ventricular tachycardia and ventricular fibrillation. Adequate oxygenation is the single greatest measure that can be used to overcome or combat these three cardiac irregularities. Procaine amide or procaine hydrochloride will aid, but we have found methoxamine hydrochloride the most efficacious drug to control ventricular tachycardia.

Shallow depths of anesthesia, adequate oxygenation, constant assays of the physiological status of the patient, and the ability to combat hemorrhage quickly are the essentials for the anesthesiologist during the course of anesthesia for the surgical correction of aortic stenosis. Excellent results can be obtained by teamwork and many patients returned to a normal life following aortic commissurotomy.

and administer 10 to 12 liters of oxygen per minute as soon as the patient has been placed on the operating room table

A muscle relaxant of rapid action, decamethonium bromide (Syncurine) 2 to 4 mg is injected intravenously, followed by 200 to 350 mg of thiopental sodium intravenously. The oxygen face mask is then removed. A large bore endotracheal catheter is introduced into the trachea under direct vision. Oxygen is administered to the patient and the lungs are inflated by rhythmically compressing the breathing bag.

It is absolutely essential to have an associate keep track of the cardiac action during induction and intubation. Ventricular fibrillation is most prone to occur in patients with aortic stenosis. If ventricular fibrillation does occur, the left chest must be entered immediately and the aortic valve dilated by means of introducing a dilator through the left ventricle and through the stenotic valve. It has not been possible to overcome this dread arrhythmia if aortic stenosis is dynamically significant prior to doing an aortic commissurotomy. Obviously this technique is not desirable but it can be accomplished rapidly and bloodlessly (due to inadequate cardiac action) with hemostasis being secured after bleeding and cardiac action have been restored.

Transaortic commissurotomy is achieved with the patient in the supine position ordinarily through a right thoracotomy incision. It is necessary occasionally, to have a bilateral thoracotomy with resultant bilateral pneumothorax and the attendant abnormal intracardiac pressure changes.

Hemorrhage can result from a laceration of the aortic wall at the location of the pouch just distal to the valve or from instrumentation through the pericardial pouch that has been sutured onto the aorta.

less than thirty years of age, free of cardiac failure, has cardiac enlargement of only moderate degree, and has a palpatory systolic blood pressure of more than 100 mm of mercury when turned onto the right side

The simple test of ascertaining the true blood pressure in the lateral position has been our chief indicator as to the ability of the heart to tolerate anesthesia and surgery

The multiplicity of proposed operations for mitral insufficiency in the past is adequate proof that none was universally accepted or successful. The mortality rates during and after surgery are adequate testimony to the validity of inadequacy. Nichols in August 1955 performed his first cross polar plication for mitral insufficiency. The series to date is small—thirty five cases—but the operative and post operative mortality rate has been only 14.7 per cent with evidence of complete correction several months after surgery. To the anesthesiologist this is most gratifying because the patient's cardiac output is improved immediately after the definitive surgery has been completed.

The anesthesiologist should visit the patient several times preoperatively, to ascertain the cardiac reserve and to make a daily blood pressure curve with the patient turned onto the right side.

Night time sedation is ordered for the hour of sleep and consists of pentobarbital sodium (Nembutal 100 mg) (gr 1½). Secobarbital sodium (Seconal) is ordered for oral ingestion ninety minutes prior to the scheduled hour of induction if the blood pressure is above 110 mm of mercury with the candidate in the lateral position. We prefer small doses of not more than 50 mg (gr ¾). Atropine sulfate is ordered for hypodermic injection ninety minutes prior to induction. Extreme caution should be observed with the use of atropine sulfate. Normally atropine sulfate inhibits vagal activity thus the pulse rate will be acceler

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In mitral insufficiency the bicuspid valve of the left side of the heart fails to close properly during systole of the left ventricle. The left auricle receives the regurgitant stream of blood resulting in atrial dilatation.

The symptoms are minimal in the early stages of the deformity but since the disease is progressive increase rapidly in severity. Fatigue due to inadequate general circulation with blood regurgitating into the left atrium is the chief single complaint. Dyspnea from increased pulmonary capillary pressure, pulmonary edema and dependent edema are the other consistent symptoms.

Physical signs are quite typical. A systolic murmur heard in the mitral area is classical. The systolic systemic blood pressure is usually within normal limits. Atrial fibrillation is common. There is a decreased intensity of the mitral first sound. The left atrium is markedly enlarged. The left ventricle is hypertrophied and dilated. The left upper lobe bronchus is usually kinked from pressure made on it by the enlarged left atrium.

The anesthesiologist in evaluating the patient preoperatively with the cardiologist and the cardiac surgeon uses the guide of the American Heart Association as to functional capacity of the patient. An ideal candidate is

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through the endotracheal tube at a rate of five liters per minute by manual compression on the rebreathing bag. The cuff of the endotracheal catheter is inflated to secure an air tight fit in the trachea. During induction, it is most helpful to have an assistant who keeps a constant check on the radial pulse and aids in securing the tube to the mouth bite and the patient's face. We believe rapid intubation to be the easiest for the patient if done without trauma.

The patient is positioned for surgery, extreme care being taken that the shift of positions be done free of rapid traction of the body. If the systolic systemic blood pressure remains at the preanesthetic level, surgery can be instituted. If hypotension follows either anesthesia induction or the change in position, place the patient immediately in the supine position and cancel the contemplated surgery.

Ordinarily oxygen alone is administered through the endotracheal tube but if the blood pressure remains at least 100 mm of mercury equal portions of nitrous oxide and oxygen are administered, with a total volume of at least five liters per minute.

Thiopental sodium (Pentothal Sodium) is added by intermittent intravenous injection as needed to provide constant hypnosis. The use of muscle relaxants after intubation is not encouraged unless there is marked resistance to respiration.

The standards of constant evaluation and good anesthesiological practice must be followed or the operative mortality will be too high and the surgical procedure needlessly discarded.

ated following its use Tachycardia shortens the period of diastole to such an extent that there is inadequate filling of the chambers of the heart specifically the left ventricle is handicapped The use of large doses of atropine sulfate in patients with dynamic mitral insufficiency will then result in two most undesirable effects tachycardia and hypotension We never exceed a dose of 0.33 mg (1/200 gr) of atropine sulfate preoperatively unless the pulse rate is less than sixty per minute

The patient must have two veins cannulated prior to the induction of anesthesia It is preferable to have the one infusion placed in the dependent arm and one in the dependent leg The blood pressure cuff and stethoscope are placed on the left arm This will not hinder the administration of parenteral fluids when the blood pressure cuff is inflated and will also allow more accurate blood pressure recordings to be made during anesthesia The stethoscope is of dubious value during the course of anesthesia when the patient has atrial fibrillation First it is advantageous to recall that the palpatory recording is higher than the auscultatory and second it has been almost impossible to obtain consistent diastolic records in the lateral position if atrial fibrillation is present, or occurs during the anesthesia

A muscle relaxant of short but rapid action decamethonium bromide (Syncurine) 2 to 4 mg or succinylcholine hydrochloride 20 to 40 mg is injected through a three way cock into the cannula in the right arm followed by 200 to 350 mg of thiopental sodium (Pentothal Sodium) into the same vein A large calibre catheter with inflatable cuff is without trauma quickly introduced into the larynx and trachea a mouth prop is inserted so that the catheter can not become obstructed by the teeth clamping on it and oxygen is administered to the patient

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Thiopental sodium (Pentothal Sodium) is added by intermittent intravenous injection as needed to provide constant hypnosis. The use of muscle relaxants after intubation is not encouraged, unless there is marked resistance to respiration.

The standards of constant evaluation and good anesthesiological practice must be followed or the operative mortality will be too high and the surgical procedure needlessly discarded.

ated following its use Tachycardia shortens the period of diastole to such an extent that there is inadequate filling of the chambers of the heart specifically the left ventricle is handicapped The use of large doses of atropine sulfate in patients with dynamic mitral insufficiency will then result in two most undesirable effects tachycardia and hypotension We never exceed a dose of 0.33 mg (1/200 gr) of atropine sulfate preoperatively unless the pulse rate is less than sixty per minute

The patient must have two veins cannulated prior to the induction of anesthesia It is preferable to have the one infusion placed in the dependent arm and one in the dependent leg The blood pressure cuff and stethoscope are placed on the left arm This will not hinder the administration of parenteral fluids when the blood pressure cuff is inflated and will also allow more accurate blood pressure recordings to be made during anesthesia The stethoscope is of dubious value during the course of anesthesia when the patient has atrial fibrillation First it is advantageous to recall that the palpatory recording is higher than the auscultatory and second it has been almost impossible to obtain consistent diastolic records in the lateral position if atrial fibrillation is present or occurs during the anesthesia

A muscle relaxant of short but rapid action decamethonium bromide (Syncurine) 2 to 4 mg or succinylcholine hydrochloride 20 to 40 mg is injected through a three way cock into the cannula in the right arm followed by 200 to 350 mg of thiopental sodium (Pentothal Sodium) into the same vein A large calibre catheter with inflatable cuff is without trauma quickly introduced into the larynx and trachea a mouth prop is inserted so that the catheter can not become obstructed by the teeth clamping on it and oxygen is administered to the patient

all other valvular defects the palpatory recording is the highest. The diastolic blood pressure is extremely low, frequently zero. These wide variations result in a wide pulse pressure that is pathognomonic of the lesion. The apex of the heart is near the anterior axillary line, as a result of the compensatory dilatation of the left ventricle. The arterial pulsations are characteristically water hammer or the so-called Corrigan pulse, this is due to the wide pulse pressure. Capillary pulsations can be observed in the nail beds if major insufficiency is present.

In fluoroscopy the aorta shows a marked expansile pulsation.

Roentgenograms show cardiac enlargement involving principally the left ventricle with left ventricular hypertrophy and dilatation.

Brachial artery tracings show the dicrotic notch to be low, slurred or absent. Typically, there is a rapid ascent to the peak of systole with an equally rapid descent to a low diastolic pressure.

Electrocardiographic tracings most often reveal sinus tachycardia with left axis deviation and left ventricular strain and hypertrophy.

The anesthesiologist in evaluating the patient with the cardiologist and the cardiovascular surgeon, uses the guide outlined by Hufnagel for the ideal candidate as (1) age less than fifty years (2) pure aortic insufficiency (3) absence of acute rheumatic fever (4) signs of progression of aortic insufficiency despite adequate medical management (5) cardiac enlargement of only moderate degree, (6) absence of intractable angina pectoris (7) absence of serious change in the ventricular conduction and (8) absence of serious renal or hepatic disease.

There is a simple test that has proven very useful to the author in addition to the ideal findings outlined above,

AORTIC INSUFFICIENCY

AORTIC INSUFFICIENCY may be a result of rheumatic valvulitis, syphilitic heart disease or a congenital malformation of the aortic valve. The defect may exist alone as a pure aortic valvular lesion or with varying degrees of aortic stenosis.

In aortic insufficiency, the tricuspid valve of the aorta fails to close properly during diastole. This allows the blood from the aorta to regurgitate into the left ventricle. The left ventricle receives blood from the left atrium at the same time resulting in left ventricular hypertrophy.

The symptoms are minimal in the early stages of the deformity but since the disease is progressive head shaking due to the forceful contractions of the over-distended left ventricle develops. Angina or chest pain is produced from an inadequate diastolic pressure restricting the amount of oxygenated blood flow into the coronary arteries at their origin distal to the aortic valve. Dyspnea upon exertion is the most distressing symptom to the patient as this becomes progressively more severe resulting in paroxysmal nocturnal dyspnea. Pulmonary edema ordinarily is not manifest until the terminal phases of the disease.

Physical signs are quite characteristic: a diastolic murmur heard in the aortic area is classical. The systolic systemic pressure is elevated above normal limits of 150 mm of mercury. The auscultatory systemic blood pressure is elevated to 30 to 50 mm of mercury ABOVE the palpatory systolic determination. THIS IS THE EXCEPTION IN ALL OF THE ACQUIRED VALVULAR LESIONS. In

hypodermic injection is ordered to be given one hour (sixty minutes) before the time of anesthesia

The patient must have two veins cannulated prior to the induction of anesthesia. It is preferable to have the infusions in place in the right arm (dependent arm) and right ankle. The blood pressure recordings and pulse rate are obtained and recorded on the anesthesia chart.

It is highly advisable to place an oxygen mask over the patient's face and administer ten to twelve liters of oxygen per minute into the mask as soon as the patient has been properly placed on the operating room table. This remains in place during the induction of anesthesia. There is no drug in medicine as prophylactic or therapeutic as oxygen with which to prevent or treat arrhythmias during endotracheal catheterization.

A muscle relaxant of rapid and short action, decamethonium bromide (Syncurine) 2 to 4 mg, or succinylcholine chloride 30 to 50 mg is injected intravenously followed by thiopental barbitol sodium (Pentothal) 200 to 350 mg by the same route. Then the oxygen face mask is removed. A large calibre catheter with inflatable cuff is quickly and without trauma, introduced into the larynx and trachea. A mouth prop is inserted so the tube will not become obstructed by the teeth clamping on it, and oxygen is administered to the patient through the endotracheal tube at a rate of five liters per minute by manual compression on the rebreathing bag. The inflatable cuff is filled with air until it is air tight in the trachea and then the endotracheal catheter is secured to the mouth block and the patient's face. During the induction and intubation it is essential that an assistant keep track of the patient's cardiac output. This can satisfactorily be achieved by constant palpation of the radial artery and with frequent blood pressure and pulse rate determinations. The total time of

in ascertaining whether or not adequate results can be obtained and that is to ascertain the diastolic level of the blood pressure then to have the patient take a deep breath and strain as hard as possible without breathing. This is the Valsalva experiment or maneuver. If the diastolic pressure reaches a normal level the patient can be expected to have an adequate result and if this simple maneuver does not produce paroxysmal dyspnea the patient has sufficient cardiac reserve to tolerate the anesthesia and surgical intervention.

Bailey and Hufnagel are both in agreement that a perfect operation for aortic insufficiency has not as yet been devised. However patients will rapidly deteriorate and die once the disease becomes progressive unless they are aided surgically.

The anesthesiologist must visit the patient several times preoperatively; this not only aids in the establishment of a proper patient-physician relationship, but it allows a much better deduction to be made by the anesthesiologist as to whether the patient can be safely carried through the anesthetic period. Furthermore a guide as to the degree of sedation that will be required for preliminary medication can more accurately be obtained.

Most patients with aortic insufficiency on whom anesthesia and surgery are contemplated have difficulty sleeping at night due to the forceful contractions of the left ventricle. We sedate them with pentobarbital sodium (Nembutal) grains $1\frac{1}{2}$ (100 mg) and meperidine (Demerol) 50 mg to 75 mg at the hour of sleep the night before the scheduled anesthesia. Secobarbital (Seconal) grains $\frac{3}{4}$ (50 mg) orally and meperidine (Demerol) 50 mg to 75 mg by hypodermic injection are ordered one hour and thirty minutes (ninety minutes) prior to the time of induction of anesthesia. Atropine sulfate grain $\frac{1}{150}$ (0.4 mg) by

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anesthesia to this point should not exceed one minute (sixty seconds)

If the Hufnagel valve is to be used the patient is turned onto the right side immediately after the positioning the blood pressure should again be determined and if it has dropped more than 30 to 50 mm of mercury (recalling that the usual preanesthetic recording was 200 to 300 mm of mercury systolic) the patient should gently but quickly be turned to the supine position and the contemplated surgery cancelled

All forms of anesthesia are depressing to the vital functions of the body and if such a light plane of anesthesia in a well oxygenated patient can not be tolerated do not subject him to the further trauma of surgery Disaster alone will follow

Annular constriction of the aorta is accomplished with the patient in the supine position Frequently a bilateral thoracotomy is necessary to visualize and dissect the right and left coronary arteries Constant evaluation of the anesthesia and the patient is necessary as any obstruction of the coronary artery will result in anoxia to either ventricle and ventricular fibrillation will ensue in a matter of fifteen to thirty seconds The anesthesiologist should stand so that he can see the heart action as well as feel a peripheral artery (usually the left radial) the first sign of danger is the occurrence of ventricular extrasystoles followed by ventricular tachycardia and then ventricular fibrillation It is not unusual to see one ventricle fibrillating independently of the other ventricle

Oxygen should be utilized the second drug of choice is methoxamine hydrochloride (Vasoxyl) 5 to 10 mg Methoxamine hydrochloride is injected into a vein almost immediately the pulse rate will become slower and the output of the ventricles will be greater It is unwise to renew the

manipulation of the major coronary artery immediately. The anesthesiologist must insist that the operator wait until the cardiac rate has been regular in force and rhythm for a time period of at least five minutes before attempting the isolation of the annulus for the second time.

The anesthesiologist working either alone, or in conjunction with a clinical physiologist, must tell the operator when the valve has been satisfactorily corrected. This is determined by the establishment of an adequate diastolic pressure, a decrease in systolic pressure, and the freedom from conduction defects to the ventricle.

The anesthetic problems most frequently encountered are (1) arrhythmias due principally to inadequate oxygenation (2) singultus from phrenic nerve or diaphragmatic irritation which we treat with small doses (25 to 50 mg) intravenously of meperidine (Demerol) (This has proven more satisfactory than curarization, regardless of the neuro-muscular blocking agent selected) and (3) hypotension, either precipitated by anesthesia overdosage, or by a failing myocardium. This responds well to a mixture of 40 mg of methoxamine hydrochloride (Vasoxyl) and levarterenol bitartrate (Levophed) 4 mg in 500 ml of 5 per cent glucose and distilled water and finally (4) ventricular fibrillation or asystole which is combatted by the usual standard methods of oxygenation, manual compression of the heart and electrical counter shock. Once ventricular fibrillation occurs in the ventricles of a patient with aortic insufficiency the prognosis is extremely poor. The ventricles are markedly atonic and hypertrophic and respond poorly to any therapy.

Patients with aortic insufficiency are the least satisfactory of all candidates for a hopeful outlook following surgical intervention.

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The selection of patients for any of the various operations to improve the circulation of the myocardium is a difficult task. Obviously, the anastomosis of the Beck-Kralick operation is a long tedious procedure. Since all forms of anesthesia are depressing to the myocardium, a patient must have a reasonable cardiac reserve to survive the anesthesia and surgery. Many post-coronary patients can not fulfill the criteria suggested by Likoff and Geckler—namely a patient of fifty years or younger who has not had a single myocardial infarction for at least six months prior to surgery, who possesses a heart of normal size, or not more than 10 per cent above normal and who is free of decompensation.

Major contraindications to surgery are a recent myocardial infarction, two infarctions within one year, congestive heart failure, hypertension, or older than fifty five years of age. There are other factors that constitute minor contraindications: such as obesity, diabetes, emphysema or renal disease.

Preoperatively the patient should remain at bed rest except for bathroom privileges. Angina pectoris is controlled by glyceryl trinitrate orally supplemented if needed, by hypodermic injections of 50 to 100 mg of meperidine.

The night prior to surgery a restful pain free sleep should be provided by ordering Nembutal 100 mg (grains $1\frac{1}{2}$) and meperidine 75 mg at the hour of sleep. The barbiturate should not be repeated later if pain awakens the candidate but the angina controlled by glyceryl trinitrate and Demerol.

Preoperatively, medication should be on the heavier side of normal as each patient fears he will be seized with angina prior to the induction of anesthesia.

Once the candidate is in the operating room, absolute quiet is essential. The blood pressure cuff and stethoscope

CORONARY ARTERY DISEASE

THE CAUSE of coronary artery disease is not established. It is however a major cause of mortality and morbidity in our present day society. Probably years of research are ahead of us before either the cause, prevention or treatment can be ascertained. Unquestionably the cardiologists and biochemists will achieve spectacular results with this disease soon. The surgeons try to salvage lives with various operations at the present although no single procedure is universally accepted by cardiologists or surgeons. The principle of all the various operations is to bring about a revascularization of the myocardium.

Claude Beck of Cleveland, Ohio, has been the pioneer in surgery for coronary artery disease in the United States. Beck has advocated several operations starting with cardio-omental pexy in which a piece of omentum was sutured to the myocardium. The original cardio-omental pexy was done by O'Shaughnessy. Another procedure advocated later by Beck was to open the pericardium and introduce an abrasive such as talc into the pericardial sac.

Beck and his coworkers found that it was possible to make an anastomosis (really an arterial-venous fistula) between the coronary sinus and the aorta reversing temporarily the circulation of the coronary system.

Thompson and Raisbeck use powdered talc in the pericardium to stimulate the blood supply to the pericardium. Vineberg implants the left internal mammary artery into the wall of the left ventricle to revascularize the heart. Murray suggests the actual removal of the thrombus from the coronary artery.

Undue pressure on the heart by a member of the surgical team will result in instantaneous hypotension. Hypotension precedes cardiac irregularities and the traction or pressure must be released by the surgeons as quickly as detected by the anesthesiologist.

Anoxia will precipitate fibrillation of the diseased ventricles in a matter of two to three minutes; thus even transitory obstruction to the airway can not be tolerated.

A mixture of equal parts of nitrous oxide with oxygen offers satisfactory analgesia. The combination of intravenous pentothal sodium and succinylcholine affords an easy achievement of inflation of the lungs. Intravenous procaine has not stood the test of time for coronary artery disease patients in assuring freedom from arrhythmias. Arrhythmias are most easily avoided by adequate oxygenation and the avoidance of deep anesthesia.

are placed in the proper position quickly and the systemic blood pressure obtained. Induction of anesthesia should be rapid. We use either 40 to 50 mg of succinylcholine hydrochloride intravenously or 3 to 4 mg of decamethonium bromide to facilitate intubation. The muscle relaxant is followed by 300 to 400 mg of pentothal sodium by the same route. A large bore endotracheal tube with an inflatable cuff is inserted under direct vision. Oxygen is then administered by manual compression of the breathing bag; the pulse is carefully checked for irregularities and the blood pressure ascertained. If no gross abnormalities are noted, the patient is positioned for surgery. Should premature ventricular systoles occur, or a blood pressure level of 20 mm of mercury less than ascertained in the supine position be found in the lateral position, the patient should not be subjected to the long anesthesia period. It is preferable to judiciously abandon the contemplated operation than to invite death. Whenever undesired changes occur, a decision must be made to either shorten the procedure, such as the installation of asbestos or talc powder onto the surface of the heart, or to cancel the case. Words are not strong enough to strengthen this stand. It simply must be realized that a danger exists for ventricular fibrillation to follow even momentary anoxia of the myocardium.

Singultus has been an anesthetic complication of frequent occurrence during the Kralick operation. The cause is due to either diaphragmatic irritation or constant pressure on the phrenic nerve. This complication can best be avoided by controlled respirations (free of diaphragmatic motion) with a depth of second or third plane anesthesia or curarization of the patient with succinylcholine hydrochloride. Meperidine 25 to 50 mg intravenously will correct singultus if it does occur.

from resistance and dead space, (6) adaptable to both infants and adults, (7) contain a means of absorbing carbon dioxide effectively, (8) method of recording inspired oxygen tension, (9) be sturdy enough to tolerate abuse and (10) noiseless in operation

Laboratory equipment will also be developed that will enable the anesthesiologist to secure blood pressure recordings immediately allow arterial oxygen tensions to be accurately recorded, afford a means of accurately determining the circulating blood volume and blood pH

Heart lung machines of the future will do much to decrease the mortality rates of intracardiac surgery. These machines will have the ability to afford a completely bloodless open heart, and possess an oxygenator that will not destroy the clotting ability of the blood or produce arterial embolization. It will be capable of being placed into circulation within five to ten minutes after the induction of anesthesia and finally allow the cardiac action to be spontaneous, following its use.

The same spirit that pioneered cardiac anesthesia and surgery will be triumphant.

FUTURE DEVELOPMENTS

ANESTHESIA for cardiac surgery has passed through its infancy and is making rapid strides toward maturity. There is still much to learn about the selection of patients, anesthetic management, avoidance of complications during anesthesia, and postanesthetic management and care.

It is hoped an ideal anesthetic agent will be discovered in the future. It should have as a minimum the following ten requisites:

1. Rapid and pleasant induction
2. Odorless and nonirritating
3. Nonexplosive
4. Ability to produce both analgesia and muscular relaxation
5. Wide margin of safety
6. Excretion from body unaltered
7. Ability to allow adequate oxygenation
8. Minute to minute control
9. Stable properties free of impurities
10. No side effects

A mechanical respirator should be developed that will meet the rigid requirements needed for cardiovascular anesthesia. It will have the following essentials: (1) volume control from 300 ml. to 8 liters; (2) controllable inspiratory pressure to 40 mm. of mercury; (3) a means of controlling expiratory pressure to a negative pressure as well as maintaining a positive expiratory phase if desired; (4) respiratory rate control from ten to sixty per minute; (5) freedom

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SUMMARY

IT IS NOW POSSIBLE to select patients with markedly impaired heart action who can be aided by cardiovascular surgery. The best means of patient selection has evolved as a joint conference with anesthesiologist, cardiologist and cardiovascular surgeon participating.

Patients with cardiac disease tolerate heavy preliminary medication and deep anesthesia very poorly.

Oxygen is the one gas that must be adequately administered to preserve cardiac action and life. Endotracheal catheters are essential to maintain a patent airway during anesthesia for cardiac surgery.

The most important person is the patient and every member of the team, physician, surgeon, anesthesiologist and nursing personnel must never lose sight of this concept. It is as the result of the concerted efforts of closely integrated teams that cardiac anesthesia and surgery has attained its present success.

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By

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